

# THE MEDICAL JOURNAL OF AUSTRALIA

VOL. I.—43RD YEAR

SYDNEY, SATURDAY, MAY 26, 1956

No. 21

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## An Address.<sup>1</sup>

### REMINISCENCES OF THE ROYAL ARMY MEDICAL CORPS IN MACEDONIA IN THE FIRST WORLD WAR.

By W. K. MCINTYRE,

*Retiring President of the Tasmanian Branch of the British Medical Association.*

THE following rather sketchy history of the war in Macedonia, 1915-1918, has been taken from my war diary with references from three books written after the war: "Salonica and After", by H. C. Owen; "The Salonica Front", by Captain A. J. Mann; "Macedonia. A Plea for the Primitive", by A. Goff and H. Fawcett, M.R.C.S.

#### The Military Situation on Our Arrival.

The military situation when the first British force landed at Salonica in October, 1915, was very unsatisfactory. Gallipoli had been evacuated. The attack on the Serbians by the Germans from the north and Bulgarians from the west had driven the Serbian army down to the northern

border of Macedonia and into Albania, and the British Tenth Division, which had been landed at Salonica and rushed up into southern Serbia, had held the Bulgar advance for only a short time, and so had to be withdrawn into the Salonica "Birdcage" defence line.

French and British divisions were withdrawn from the western front and transported by sea to Salonica. Many German submarines were in the Mediterranean, and a number of our ships were sunk. A submarine surfaced near the ship I was on, but we plugged it with our eighteen-pounder and zig-zagged off to safety. Others were less fortunate.

When we first landed in Salonica, Greece was still neutral, supposedly friendly, but actually engaged in active organized opposition. King Constantine's wife was the Kaiser's sister and was very definitely pro-German. He and many prominent Grecians in Athens did all in their power to join up with the Germans, and opposed all French and British movements in the Balkan area. Strictly of course we had no right to land in this so-called neutral country. What finally prevented the King and Parliament from joining the Germans was the strong attitude taken by our side later in 1915 in the following way: (i) Blockading all Greek ports by our warships, which was begun in December, 1915. (ii) Agreement with the Salonica Greek army, which allowed free circulation of our troops in Macedonia; this was due to Venizelos, who was strongly opposed to Constantine and Germany. (iii) Expulsion

<sup>1</sup> Read at the annual meeting of the Tasmanian Branch of the British Medical Association on February 25, 1956.

from Salonica of all enemy consulates immediately after the first German air-raid on the city. (iv) Our seizure and occupation of all the Greek forts round Salonica harbour.

When we landed we found ourselves in a web of hostile influence which hampered our movement in every direction. Everything we did was known to the Greeks. The town swarmed with German agents and spies, and conditions were complicated by the fact that Greece was supposedly neutral and our Army Commander, General Sarrail, had not a free hand to deal with them. However, when the Germans began the air-raids, Sarrail was able to take a strong hand.

In October, 1915, Venizelos left Athens secretly and came to Salonica. Under his influence the Salonica Greek army eventually became anti-Constantine, and the King was forced to agree to a strictly neutral attitude; eventually he had to leave Athens, and went to Switzerland in June, 1916.

The Serbian army, which had suffered terribly during the winter retreat through to Albania, had been reconstituted on Corfu Island; and as Constantine refused passage of this army across Greece, British and French naval forces brought them round by sea and landed them in Salonica.

By the end of 1915 British and French forces had consolidated their position in the so-called "Birdcage" defence line, and we were expecting the enemy to attack at any time to try to drive us out of Macedonia. The defence line stretched for about sixty miles across country from the marshlands of the Vardar delta, along the northern slopes of the Derbend range, to the western shore of the Gulf of Orfano. We soon had the greater part of this defence line reinforced by an elaborate system of concealed trenches, concrete gun emplacements and wire entanglements. We also constructed a solid metal road into Salonica, which was about eight miles south of the line. When we were digging in, the Bulgars were doing the same along the northern mountain border, from the Rupel Pass, which Constantine had handed over to them, across to Albania, and the attack we were expecting day after day, and looking forward to, never eventuated. It was ourselves who, many months later, advanced and attacked them in their impregnable position on Petit Couronné and Pip Ridges, of the Belashitzza Ranges, without success, and with very heavy losses.

The enemy air forces in the earlier part of the campaign were much stronger than our own, and we had to put up with daily bombing raids on our front lines, camps and lines of communication and on Salonica itself. Zeppelins came over frequently until the warships in the harbour brought down one on the Vardar marshes early in 1916.

#### History of Macedonia.

It may be not out of place to say a very few words about the history of Macedonia.

Cassander married a sister of Alexander the Great. After Alexander's death, Cassander, wishing to possess himself of a portion of the rich empire conquered by Alexander, ravaged Macedonia. About 315 B.C. he founded the city of Salonica, which he named Thessalonica in memory of his wife, and protected it by strong walls. It became the centre of great commercial activity, and down the centuries has been the goal of all attacks on Macedonia, because it is the only suitable port on the Macedonian coast for trading.

So Macedonia over two thousand years ago was going through the same process of recurrent invasions, as had continued to the time of which I am speaking. Costumes and weapons of the ravagers had changed, but their methods had not changed very much. Fire, pestilence, famine, earthquakes, revolutions, war and massacre, these Macedonia had known time and time again. In 1917, while we were there, they had the great fire which rendered 100,000 people homeless, with a total damage estimated at £40,000,000.

After a four-year siege it fell prey to the Turks, who in 1387 had already overwhelmed the whole of the Balkans and had the Eastern Empire in a state of vassalage. It was under the rule of the Turks for centuries until the first Balkan war in 1912. The Macedonians suffered the cruelties, oppression and torture inevitable with the religious fanaticism of the Near East—Mohammedism versus Orthodox Christianity.

The Balkan League formed by the coalition of Greece, Serbia, Bulgaria and Montenegro was finally successful (1912) in driving the Turks back to Constantinople, but the League did not hold together, owing to rivalry over Salonica. The Greeks got in a day or so before the Bulgarians, and the ill feeling culminated in the second Balkan war, when Bulgaria attacked Greece and Serbia. However, with help from Rumania the two allies beat Bulgaria, and the Treaty of Bucharest was forced on her.

Macedonia had thus been a buffer State between the east and the west over the centuries, and the ignorant Macedonian races, accustomed to most harsh and inconsiderate treatment from man and Nature, had developed a hardihood and resignation to suffering.

Owing to its being surrounded by many countries of different nationalities, the population may be described as heterogeneous. For centuries this land had been a common arena for the settlement of disputes between the Serbs, Bulgars, Turks and Greeks, and a hybrid race had thus inevitably been produced.

In spite of this intermingling, however, racial hatred was predominant, and this had been largely fostered by that insuperable gulf, the difference of religion.

Macedonian natives were ignorant and fatalistic, and remained unmoved by calamitous circumstances or overwhelming catastrophes. Also, they regarded strangers with scarcely veiled suspicion. In many ways they had probably changed but little since Biblical days, and the country villages were much the same as they were in A.D. 53, when Saint Paul travelled on his evangelical mission from Asia through Macedonia to Athens, where he preached from the Areopagus, below the Parthenon, to the Greeks.

#### Physical Geography.

Macedonia is separated from its neighbours by mountain ranges up to 5000 feet high, and Macedonia itself consists of a series of bleak mountain highlands and plains, interspersed occasionally by lakes, and everywhere else cut into by countless intersecting torrential nullahs, which rendered wheel traffic extremely difficult even in fine weather.

The two main rivers are the Vardar, which rises in Serbia and enters the Mediterranean at Salonica, and the Struma, which rises in Bulgaria and flows into the Gulf of Orfano.

When we began operations in Macedonia there was only one existing highway that could be called a road. This was the old Roman road from Durrazo on the Adriatic coast, across to Monastir, Salonica and Kavalla and ending at Constantinople. When we began to use this road, constant subsidence of the soft underlying strata made it necessary to reconstruct the whole road and constantly repair it throughout the whole campaign. The only other so-called roads from Salonica were to Naresh and Seres. Both were in a shocking condition, and quite unfit even for light traffic, let alone our heavy vehicles. Up to our arrival, in all previous wars, they had had to bear only animal traffic.

#### "The Birdcage."

During the early months after our arrival, we were doing a colossal amount of digging for trenches and defences along the steep line of hills covering the town—the Derbend Range, which was up to 4000 feet at Mount Hortiach—and also constructing roads from the city to the front line, which was about eight miles. When in 1916 we moved up country, all this trench work went to naught. The next big job was to convert the 70 miles of semi-track that ran up and down the steep hills and nullahs as far as



the Struma Valley, into a road that would take all the heavy traffic necessary to supply several of our divisions. There were steep rises up to 1800 feet and down again, and in bad weather the road simply disappeared and became a series of mud slides; the lorries would often sink into the mud up to the axles, or roll over onto their sides. Then mules took over and struggled on with what rations *et cetera* they could deal with. It was impossible to carry on in this way; so dozens of steamrollers and stone-crushers and other machinery were brought out from England as quickly as possible, and a huge army of native men and women and our own soldiers set to work (by night using acetylene flares). By the middle of 1917 the roads were completed, but still required constant repairing, especially in winter, for the rest of the campaign.

From the first to the last we took over, constructed and kept in repair 270 miles of metalled roads and another 170 miles of secondary roads, and General F. Milne, our commander-in-chief, said that "for the past two years of the campaign in Macedonia communications were the main difficulty, but like the work of the Romans of old, the roads of the British Army in Macedonia will long remain the best memorial of its presence".

#### Natives and Villages.

Time had stood still in the more remote corners of Macedonia. The native was a tiller of the soil and tilled it and threshed its products in much the same way as they did two thousand years ago.

The villages were isolated from each other, and the natives were very primitive. The inaccessibility of the interior was one of the main causes of this isolation, as were also the mixed nationalities and religious denominations, so inherently hostile, as the Mohammedan Turks and Orthodox Greeks and Bulgars. Often the villages would comprise all of the various social and religious denominations which are to be found in Macedonia, each clan living in its own quarter. Every village, even the smallest, had its own place of worship. The Turkish mosques always had the minaret varying from 20 to 100 feet in height. These were built of rough stone, and enclosed a spiral staircase, which led to the gallery on top; from there the *muezzin* called the faithful to prayer at sunrise and sundown.

Each village was a community in itself. There was no controlling authority, such as a council, and most of the villages were presided over by a headman, the village priest or the oldest inhabitant, who acted as an intermediary between his and neighbouring villages.

Constant wars and repeated local disasters down the ages had combined to bring about a "laissez-faire and let us alone" attitude. Under an overwhelming catastrophe the native would merely shrug his shoulders, or hold up his hands in impotent resignation or grief. He lived in the land and by the land; he very seldom bought food, even if he had the money to do so. He relied for sustenance on what he actually cultivated; and after he had paid his tax, by heavy contribution of his products to the ruling powers, he had not much left for himself and his family. Except in a few of the larger towns there were no banks where he could deposit money, even if he had it, and also the general lawlessness of the country would make him fear that, even if he did save and bank money, someone else would enjoy the fruits of his toil and thrift.

No generation had been immune from war and massacre, and probably no individual had been spared the sight of bloodshed. All natives had probably inflicted or received bodily injury, and witnessed the horrors of rapine.

The Orthodox Christians in Greece had a curious custom in connexion with their burial rites. The graves were shallow, and the same ground was used over and over again. Relatives exhumed the bones and placed them in a bag with the name of the deceased and the sign of the crucifix embroidered on it. This bag was then placed in a chapel adjoining the church. This practice was due to their desire to have something preserved to perpetuate the memory of the deceased. I wandered about a cemetery

in Larissa, south of Mount Olympus, and watched relatives digging up bones, some of them arguing about whether, for instance, a skull was really the dead relative's skull, and getting quite worked up. They would clean out the eye sockets, mouth *et cetera* and hand it round for inspection. I had a look in the chapel, which was a very gruesome sight; enormous piles of human bones were stored there.

#### Climate.

The Vardar wind is another of the local scourges: a terrible wind which sweeps across the Vardar marshes. It is the modern name for the old "Boreas", which was worshipped by the Ancient Greeks at Athens, because it destroyed the ships of invading Persians in the Persian War, while Leonidas was holding the Persian army up at Thermopylae.

In the summer, in July and August, when there is little rain, the Vardar wind will suddenly stir up, and instead of lowering the very oppressive heat, rather make it worse. The heat seems to increase, but its especial curse is the fine, gritty dust, which covers the face and fills the eyes, nostrils and ears; so that with every breath the grit gets into the mouth. This wind often is followed by a sudden drop in temperature, and this is followed by heavy hailstorms. Over and over again our tents would be blown away or torn to ribbons by the cyclonic force of the wind.

In the winter months the Vardar wind usually lasts for days, accompanied by piercingly cold blizzards, with temperatures well below freezing point; and apart from the physical discomfort of tent life in such weather, it was very easy to lose one's way when outside the camps travelling over rough country with few or no tracks.

#### Cultivation.

Cultivation methods were very primitive. Tools and methods had not changed much for thousands of years. Among the hill folk, every tiny bit of soil, lying amidst rock-strewn ground, was turned to some use and cultivated.

Ploughs consisted of a long beam of wood, at the end of which was a block of wood, which was set vertically and carried the iron cutter. Yoked oxen, separated by the shaft, were harnessed to it by ropes or leather thongs and dragged it slowly along. The ground was pierced only a few inches.

The British introduced motor ploughs and sowing machines, and in 1918 placed large areas in Dorian and Struma under cultivation. Results were very disappointing, owing to the ignorance of the local climatic conditions and disregard of native knowledge and customs. While native corn was almost ready for the first harvest, acres of English-sown wheat were still green or scarcely showing those young shoots soon to be caught by a blazing sun.

It is interesting that in some schools of modern agriculture there is a tendency to believe that it is better for the soil not to be deeply disturbed.

Threshing and winnowing processes were very interesting to watch. Every village had its own circular, flat threshing floor, with a flat, well-beaten, hard and smooth surface. The corn was laid on it a few inches deep, and large sleighs of wood, like flat toboggans with sharp projecting stone splints set underneath, were dragged round and round by oxen or donkeys. The splints were sunk in cuts in the under surface of the sleigh, which had been swollen in water, and were firmly fixed when the wood dried. The flints were the result of clever chipping, and were identical with the flakes used by prehistoric man before the Bronze and Iron Age. The grain was thus partially separated and shelled out. Heavy marble rollers were then run over it, after which the grain was collected and the straw was stacked in heaps. Winnowing was carried out by the simple method of throwing the grain in the air by wooden scoops when there was a suitable breeze; the chaff was blown to one side, while the grain fell down onto the floor. The grain was ground with stone pestles and mortars worked by hand, or in some villages by small watermills. The miller was paid by being allowed to keep a percentage of the flour he produced.

### Crops for Home Consumption.

Wheat, mealies, oats and rye were grown for home consumption, and the villagers might get two or even three crops a year on those fertile plains.

Another crop for home use was cotton. This was grown by the villagers for their own use, and the women spun it to make their own garments with it.

Rush mats were made from big reeds from the swamps. These mats were used by the natives for roofing, floors and sun screens.

### Crops for Export.

Two of the main crops for export were tobacco and opium.

Tobacco was foremost on the list of industries. It was seen all over Macedonia, and the plains of Drama produced the finest oriental tobacco in the world. The leaves were picked and hung on verandas and mud walls of the houses in the sun. When brown and dry, they were packed in bundles, pressed and left for months in the dark to ferment. Finally, when the leaves were dark, soft and sticky, they were ready to be cut into shreds and exported. Kavalla was the chief town for its export.

Grown for the production of opium, huge expanses of white poppies might be seen, when they were in full bloom, on many of the plains of Macedonia. When the petals fell off, green seed pods remained on the stalks. These pods were scratched three-quarters of the way round, with a double-pronged instrument. A thick sticky juice exuded, milk white at first, and then brown. Tiny drops formed and were collected every few days, and this was repeated until the seed pod dried up. The sticky mass was dried, moulded into cakes, which became black and tarry, and then wrapped in poppy leaves and packed for export.

### Women's Work.

Macedonian women had a pretty tough life. Having been under Turkish rule for several centuries, the Greek Orthodox men treated their women the same as the Mohammedans, and kept them well under their thumbs.

The wife had to do her share of outdoor work (planting, harvesting *et cetera*) as well as the domestic work.

They matured earlier than Western women, and usually married in their early teens, having their first baby at about fourteen years of age. By the middle twenties they had a number of children and became rather obese and looked old. They carried on with their manual labour up to a day or so before confinement, and returned to work, in most cases, one or two days after the birth.

Children at a very early age had to help both in and out of the house, so that they had not the same childhood as the Westerners have.

When going out to the fields the father, who was well shod, rode the donkey, and the women and children, barefooted, trudged meekly behind, usually loaded up with clothes and equipment.

The Greeks had no sympathy for suffering animals. They grossly overloaded their thin wretched-looking donkeys, and we often found badly injured animals abandoned by the roadside, which we always put out of their misery with a bullet.

### Transport of Sick and Wounded.

This rather sketchy description of the local conditions may emphasize some of the difficulties the field ambulance had to contend with in transferring sick and wounded to base hospitals.

Our first problem after landing was to transfer all our ambulance equipment so that it could be carried on pack mules.

In the Twenty-eighth Division two Australians and one New Zealander were transferred from Medical to Army Service Corps work. The late Dr. B. M. Carruthers, our late Director of Medical Services in Tasmania, took over the Seventy-eighth Field Ambulance Section, Lieutenant

Davie, who recently died in New Zealand after attaining a high reputation in the College of Surgeons, took over the Seventy-ninth, and I took over the Eightieth.

Thousands of mules were brought over from South America. Some were broken into pack work, but many of them were not, so we had to train them. We set to work at once, changing the equipment for carriage by the mules, not an easy job, and training the men in loading and unloading, and also breaking in the mules. We were expected to know more about mules and packing than the English and Scottish laddies.

While this change was going on, the immediate and more urgent problem had to be solved of dealing with sick and wounded, and of getting them down from the front lines to aid posts, and then on to where the ambulances took over. This we found particularly interesting, and we invented many different types of conveyances that could be used on the tracks as well as over trackless country. We and our carpenters constructed and presented them to the Assistant Director of Medical Services and other high officials for condemnation or approval. If they thought that the conveyances were worth a trial, practical tests were carried out over difficult country to prove their worth.

The old American Indian travois, a primitive vehicle of two trailing poles, serving as shafts, from which the forward end of the stretcher was suspended by ropes, the other end resting on a cross-bar, proved to be, perhaps, the most generally useful conveyance. We found that having one of the travois shafts longer than the other prevented a lot of jolting over rough, rocky ground. The front of the stretcher being suspended by ropes, this prevented jolting at that end. A minor trouble was that the patient's head was rather close under the mule's tail, with obvious consequences now and then.

Cacolets had seats on each side suspended from the saddle and allowed two not severely wounded or sick men to be carried by one mule. Monowheels, litters on the mule's back or suspended between two mules, were constructed and tried out, reported on and used by various field ambulances. The travois and cacolets were the most used, especially the travois, which proved a very comfortable conveyance, even on the shockingly rough tracks we had to use.

### Malaria and Other Diseases.

There had been an epidemic of cholera just before 1914 in the Near and Middle East, which fortunately had subsided; and though we were on the lookout for a possible recurrence, we had no cases.

Epidemics of dysentery occurred at intervals and caused us some worry on the front lines in the earlier months owing to the possibility of a recurrence of the recent cholera epidemic.

Typhus had played havoc with the Serbian army during and after their retreat, and we took special precautions by routine examination of all units for body lice. Any men found, when lined up, to have skin scratches on their bodies were immediately sent down to the delousing camp, where all garments were deloused before they returned to their units.

Malaria, of course, was the main curse. The number of admissions to hospital for malaria alone in the summer of 1916 was just under 30,000. Many of these patients had to be brought from the front line on travois and cacolets, and the field ambulances had a very exhausting time coping with them. The total number of admissions for malaria increased each summer; in 1917 malaria admissions totalled 63,000, and in 1918 they totalled 67,000, from a much-depleted army, which by this year was full of listless, anæmic and hollow men, whose lives were a physical burden to them. They circulated backwards and forwards between hospital and convalescent camps. Then, when they had returned to their units, in a few weeks they were sent back on the same round again.

I got mixed up with all this work when I was appointed one of the malaria officers to the division in 1916, to organize preventive measures. Practically everyone in the



army sooner or later developed malaria. Preventive measures on a large scale were carried out, such as draining, and oiling stagnant pools, construction of mosquito-proof huts and provision of tents, gloves, headnets and various ointments. The large lakes, such as Langaza and Doiran, were quite impossible to deal with. Units were lined up and quinine was served out as a prophylactic; it was the only drug for malaria we had in those days, but it was not much help prophylactically.

Sir Ronald Ross came out in 1916, and again in 1917, to report on, and to try to improve the situation. On his second trip the ship on which he was crossing from Taranto, in the south of Italy, in the Gulf of Corinth, was torpedoed, and he lost all his luggage.

My friend Lieutenant Davie, of the Seventy-ninth Field Ambulance, and I had been on a week's leave in Athens in 1917. On the way back in a second-class carriage (the first-class carriages were full of French officers) we stopped at Bralo station, where two officers in captain's uniform got in and sat opposite us. One of them was very annoyed that British officers should be travelling second class, and considered it an insult to our army, and rather blamed Davie and me for putting up with it. Personally we did not care, as they were all pretty filthy. Then he said that he was in a borrowed uniform, and we did not care much about that either! A Greek then came along and took him out, and the other chap said: "Do you know who that is?" We did not, of course; so he then broke it gently that he was Sir Ronald Ross, who had lost all his equipment and clothes on the torpedoed ship. A few stations on he sent for Davie and me, and gave us a seat in his first-class carriage. A little farther on the carriages broke down, and we were all transferred to an open truck, on which we finally arrived safely back in Salonica.

Ross was very inquisitive about the conditions at the front, and asked us many questions and for our opinions on the anti-malarial organization, which we answered frankly. Before leaving the truck, he took our names and units, and later I wondered whether we might get into a bit of hot water, if he passed our criticisms on to headquarters.

After his second visit, the so-called "Y" scheme was brought into operation, by which all chronic malaria patients were sent home, and from January to December in 1918, 30,000 of these were evacuated.

In the summer, flies were almost more trouble than mosquitoes. They swarmed over the camps, and we tried ways of dealing with the animal manure, collecting and covering the heaps with earth *et cetera*, with little success. At meals in a tent one had to keep one's hand waving over the plate to keep them off, and then they would settle on the food in the spoon on the way to the mouth. We had fly papers at times in the tents, but one application on the tent wall would "fill it", so we soon ran out of them.

#### 1918 Influenza Epidemic.

In August, the severe attack of influenza started in Macedonia, and, as we will remember, eventually spread round the world. It reached us as we were making ready for the final assault on the Bulgar lines, and, during the attacks and the pursuit, added greatly to field ambulance and hospital work.

General Milne's dispatch in December, 1918, stated that our fighting strength had fallen below one-half of the normal establishment. The influenza spread with almost explosive force, as an official report stated, and during September and October there were nearly 12,000 admissions to hospital for influenza, over 1000 with pneumonia. The mortality in these cases was very high, as would be expected, as most patients had the prevailing debility following malaria.

#### Work Done by Women in Hospitals and Other Places.

Captain Mann, in his book, "The Salonica Front", stated that the efforts of our Medical Service, and particularly their organized sanitary and anti-malarial campaign, alone enabled the comparatively few British officers and men

who remained to retain sufficient health to carry on duty to the end.

The devotion of certain British men and women doctors and nurses up country, ending fatally for themselves in many instances, is among the most outstanding of individual efforts of the whole campaign. To all who saw the excellent work done by women in Macedonia, The Scottish Women's Hospital, the only British unit then controlled entirely by women, cannot fail to record vivid memories of an efficiently managed and very nobly conducted organisation. Nor should the self sacrifice of those women, who here or elsewhere, volunteered as motor drivers, and nurses up country, be omitted from any account of women's effort in the war. A few served for long years attached to the Serbian Army.

#### Fighting in the Balkans, 1915-1918.

There were four main operations.

First was the Franco-British expedition in 1915 to try to save Serbia when she was attacked by the Germans from the north and Bulgarians on her right flank. The British Tenth Division got up to southern Serbia, where the Bulgars had driven the Serbs almost to the Macedonian border, and had some severe fighting, but had to fall back on Salonica; we dug in the "Birdcage" defence line, expecting to be attacked any day, but determined that they would not succeed in driving us out of Macedonia.

In the summer and autumn of 1916 the French and British forces advanced from the "Birdcage" defence line and made heavy attacks on the Bulgar trenches on the Doiran front; but the Bulgars attacked the Serbs on our left and drove them south as far as Lake Ostrovo, and our attack was stopped, as we had to send reinforcements to check this advance on our flank.

In 1917 we again began an offensive on the Bulgar trenches on the Pip Ridge which were practically impregnable. We had very heavy losses and had to fall back on our trenches. The weather was extremely bad, as the attack was begun too early.

By 1918 our forces had increased even though the general health of the army had deteriorated, owing principally to malaria. New Greek divisions were rapidly coming into line and were taking over sections of the front line. The Struma Valley from Orfano Gulf to Sereas had been held by our Twenty-seventh Division for two and a half years. This was now taken over by the Greeks, and the Twenty-seventh Division moved to the left and took over the Vardar trenches. My division, the Twenty-eighth, moved west to a shorter line near Lake Doiran, and our four British divisions were concentrated on a front of 35 miles. The Cretan Division joined up with our Twenty-eighth, and later I and my B Section were transferred to this Cretan Division as the Greek medical services were very primitive, and we had many difficulties to contend with, not the least being the language. I had an interpreter attached to me, but he was not much good.

The French and Serbs with some Greek divisions occupied the rest of the front to Albania. In April and May raids on a large scale began all along the front from Doiran to the Struma to keep the enemy occupied, while the Serbs and French made ready for the main attack in September, on the less mountainous western front.

In all we had 28 infantry divisions, eight French, nine Greek, six Serbian, four British and one Italian, when we began the final operation on September 15. The success depended on whether the French and Serbs could break through a gap and split the enemy forces. Unless they could do so, we had little hope of succeeding. We had twice failed to do the impossible and drive them out of their impregnable positions on the central mountain ranges. Our task on the rest of the front was to hold the enemy from withdrawing part of their army from our front to reinforce their western front; and this we did, but suffered very heavy casualties. Our Argyle and Sutherland Highland Regiment suffered 75% of casualties, and two other of our Scottish battalions suffered 50% each; other divisions suffered similar heavy losses.

By September 21 the Serbs had broken through and cut the enemy communications on the Vardar. By September 22 the enemy began their retreat, the pursuit began along the whole line, and we slowly pushed forward. Our aeroplanes exacted a terrible revenge on the retreating Bulgars till the Armistice on September 29, when they capitulated unconditionally. Eventually after several weeks we reached the Danube, and our war was over.

As General Milne stated, "this crowning achievement after a long wearying vigil in a secondary theatre of operations struck at the Achilles heel of the Central Powers, and materially aided in their rapid collapse during the dramatic Autumn of 1918".

Ludendorff himself after the war stated: "August 8th was the black day of the German Army in the history of the War. This was the worst experience that I had to go through except for the events that from September 15th onwards took place on the Bulgarian front and sealed the fate of the Quadruple Alliance. It very soon became clear that from Bulgaria nothing more was to be expected. The position in the field could only become worse. It was impossible to tell whether this process would be slow or precipitate. The probability was that events would come to a head within a measurable time, as indeed actually happened in the Balkan Peninsular and on the Austro-Hungarian front in Italy. In this situation I felt incumbent upon me the heavy responsibility of hastening the end of the war and of promoting decisive action on the part of the Government."

Hindenburg, speaking of the Bulgarian collapse in his famous letter, said: "It is no longer possible for us to resist; we must ask for an armistice."

These quotations prove, I think, that our work in Macedonia over three long weary years had a definite value in the ultimate defeat of the Central Powers.

#### TEN YEARS' OBSTETRICS IN A COUNTRY PRIVATE HOSPITAL.

By PAUL HOPKINS,  
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THIS report presents the results of all obstetric cases conducted at the Mater Misericordiae Private Hospital, Mackay, during the ten-year period from January 1, 1945, to December 31, 1954.

Owing to the generous cooperation of all the medical practitioners concerned and also of the hospital staff, there has been no selection of cases. The 4652 cases here reported represent all of the confinements conducted at this hospital during the ten-year period which is under consideration.

#### Parity and Methods of Delivery.

There were 4652 patients confined; of these 1474 were *primiparae* and 3178 were *multiparae*. Thirty-one sets of twins were born; 14 of these were born to *primiparae*.

The total number of babies born was 4683; 4606 were born alive, and of these 4533 survived the neonatal period.

The 1474 *primiparae* were delivered of 1486 babies; of these 1456 were born alive and 1432 survived the neonatal period.

Table I shows the numbers for the various methods of delivery for all patients and also for the *primiparae* alone, together with the numbers of stillbirths and neonatal deaths for both categories for each method of delivery.

This table will repay careful study. In the normal vertex deliveries the stillbirth and neonatal death rates are greater among *primiparae* than in the whole series; in the other three methods of delivery this is not so. The stillbirth and neonatal death rates for the instrumental and Caesarean deliveries among *primiparae* are essentially the same; this may be expected in the Caesarean

deliveries, but in the instrumental delivery group a rise in the *primipara* infant mortality rate would surely be expected.

The instrumental results would be influenced by the practice, in *primiparae*, of applying forceps when the head is crowned and lifting it over the perineum. This would raise the instrumental delivery rate among *primiparae* without affecting the neonatal stillbirth rate. This claim is sustained by the instrumental delivery rate, which is approximately 64% for *primiparae* and approximately 31% over all cases.

The breech deliveries are surprising, in that the stillbirth and neonatal death rates are appreciably lower among *primiparae* than they are in the whole series.

Two reasons may be suggested for this result in the breech deliveries: one is the more careful ante-natal supervision of *primiparae*; the other is that more active methods of treatment are adopted for *primiparae* with breech presentations, with a more careful search for the cause. This would take some of these cases into another category; for instance, in four cases of breech presentation in *primiparae* delivery was effected by Caesarean section, and some others would have had Caesarean section for disproportion or *placenta praevia* (Table I).

#### Twins.

Thirty-one twins were delivered; 14 of these were born to *primiparae* and 17 to *multiparae*.

Three babies were stillborn. One had the cord tightly round the neck, one had been macerated for some time and one died *in utero* during delivery. Three babies died in the neonatal period; one died of a birth injury; two (both of the same delivery) died of prematurity (six months). Two stillbirths and one neonatal death occurred among *primiparae*; and one stillbirth and two neonatal deaths (the twins who both died) occurred in *multiparae*.

In one case both babies developed haemorrhage of the newborn, and in another case one of the twins developed *icterus gravis* and the other did not. All these babies recovered.

#### Caesarean Section.

The rate for Caesarean section for this series is 2.4%, there having been 111 Caesarean operations; 37 of these operations were performed by classical technique, and 74 were performed by the lower segment technique.

The mortality figures show two maternal deaths, three stillborn babies and five neonatal deaths.

Of the two mothers who died, one was admitted to hospital in eclampsia, showed no response to treatment and continued to have convulsions after operation; the other death was due to a sudden pulmonary thrombosis in a patient apparently doing well after operation.

Of the three stillborn babies, one died *in utero* three weeks prior to operation, the mother having shown no response to various methods of induction of labour, and one died as a result of severe eclampsia in the mother; the third patient who had a stillborn baby was a *primipara* with a transverse lie and severe toxemia.

Of the babies who died in the neonatal period, one had *spina bifida*, two were premature (seven months) and an operation for *placenta praevia* was performed, one died from *icterus gravis* in 1948 and the last died from hyaline membrane.

The reasons for Caesarean operation are shown in Table II. In the 55 cases of disproportion, 23 patients were given a trial of labour.

A study of Table III shows a marked improvement in all the figures in the last five-year period compared to the first. This improvement is almost certainly due to the following factors: (i) early and more rigorous treatment of toxemia; (ii) readily available intravenous therapy, including an excellent blood bank; (iii) the earlier decision to perform Caesarean section. In earlier years Caesarean section was at times a rather desperate remedy for a



TABLE I.  
Methods of Delivery.

Method of Delivery.	All Patients.			Primiparae.		
	Total Number of Babies Born.	Number of Stillbirths.	Number of Neonatal Deaths.	Number of Babies Born.	Number of Stillbirths.	Number of Neonatal Deaths.
Normal vertex .. .. .	2967	31 (1.0%)	46 (1.5%)	447	12 (2.6%)	11 (2.4%)
Breech .. .. .	131	22 (16.0%)	8 (6.1%)	39	5 (12.8%)	0
Instrumental .. .. .	1474	21 (1.4%)	14 (0.9%)	960	13 (1.3%)	10 (1.0%)
Cæsarean section .. .. .	111	3 (2.7%)	5 (4.5%)	40	1 (2.5%)	2 (5.0%)
Total .. .. .	4683	—	—	1486	—	—

cause which had already been lost. The simplification of intravenous therapy and the availability of antibiotics have made the decision to operate easier.

However, it must be emphasized that this factor has not been abused, as it will be seen from Table III that in 1953 the Cæsarean section rate was 1.7% and in 1954 it was 2.7%.

#### Maternal Mortality.

There were eight maternal deaths in this series, and 4606 live births.

The rate per 1000 live births was 1.73.

The Queensland figures for the same period average 1.41 per 1000 live births.

TABLE II.  
Reasons for Cæsarean Section.

Indication for Cæsarean Section.	Number of Cases.
Disproportion .. .. .	55
Placenta prævia .. .. .	21
Transverse lie .. .. .	7
Toxæmia in mother .. .. .	11
Dead fetus; all induction failed .. .. .	1
Mother gross arthritis and deformity .. .. .	1
Breech presentation in <i>primipara</i> .. .. .	4
Gross obesity with pendulous abdomen .. .. .	2
Previous repair operation .. .. .	3
No reason given .. .. .	1
Prolapse of cord .. .. .	1
Prolapse of cervix .. .. .	1
Previous classical Cæsarean section and myomectomy .. .. .	1
Acute appendicitis at term .. .. .	1
Advanced age in <i>primipara</i> .. .. .	1
Total .. .. .	111

In 1953 and 1954 there were no maternal deaths in this series, and 1138 living babies were born during these two years.

#### Analysis of Maternal Mortality.

**Toxæmias.**—The deaths from toxæmia occurred as follows. One patient had severe eclampsia, continued to have convulsions after Cæsarean section, and showed no response to treatment at any stage (1947). Another patient was a chronic nephritic; she was admitted to hospital at the eighth month for treatment for toxæmia, had a surgical induction of labour after a few days, and died of renal failure (1949). The third patient had post-partum eclampsia; the toxæmia responded well to treatment, but she died of a cerebral accident sustained during the first convulsive seizure (1952).

**Shock and Collapse.**—The death due to shock and collapse occurred after a prolonged labour with a living child; the mother went into shock and collapse, and died in spite of intravenous therapy (1952).

**Pulmonary Thrombosis.**—Two deaths were due to pulmonary thrombosis. One mother died shortly after a prolonged labour with a stillborn child (1945). The second death was due to a sudden attack in a mother apparently doing well after Cæsarean section. The baby died a few days later of *icterus gravis* (1948).

**Exhaustion and Myocarditis.**—The death from exhaustion and myocarditis occurred as follows. The patient was a small, poorly developed girl with a damaged heart. Spontaneous labour occurred at seven months; the baby weighed one pound six ounces, and died in one hour (1950).

**Puerperal Insanity.**—One mother committed suicide twenty days *post partum* whilst undergoing treatment for puerperal insanity (1951).

**Comment.**—In the first case in which maternal death followed toxæmia, earlier diagnosis and treatment may have saved both mother and baby; but in 1947 it was difficult to persuade women, particularly from the country, to come in frequently for ante-natal supervision. The first patient who died from pulmonary thrombosis would

TABLE III.

Year.	Number of Cases.	Cæsarean Operations.	Stillbirths.	Neonatal Deaths.	Maternal Deaths.
1945	308	4 (1.2%)	0	1	0
1946	345	3 (0.8%)	1	0	0
1947	412	9 (2.1%)	1	1	1
1948	438	16 (3.6%)	1	1	1
1949	471	10 (2.1%)	0	1	0
1950	478	13 (2.8%)	0	0	0
1951	512	13 (2.5%)	0	0	0
1952	548	17 (3.1%)	0	0	0
1953	528	9 (1.7%)	0	1	0
1954	612	17 (2.7%)	0	0	0
Total..	4652	111 (2.4%)	3	5	2

certainly have a Cæsarean section today; but in 1945, before antibiotics were available, the decision was in keeping with the practice at that time. Of these babies, three were stillborn; two died in the neonatal period, one from *icterus gravis* in 1948. Three babies survived. Four of these patients were *primiparae* and four *multiparae*.

#### Stillbirths and Neonatal Deaths.

Table V shows the stillbirths and their causes, and also those of the first three years and the last three years, together with the numbers of cases involved.

Table VI gives similar figures for the neonatal deaths which occurred.

Table VII shows the total mortality for each year, the number of cases involved and the rate per 1000 births. This shows considerable improvement, with a bad period in the centre—1949 and 1950.

Table VIII shows details of stillbirths for each year.

Table IX shows the details of neonatal deaths for each year with the degree of prematurity of the infant.

#### *Causes of Stillbirth.*

**Prolapsed Cord.**—There were 24 cases of prolapsed cord; 13 infants lived and 11 were stillborn. Eight cases occurred between 1945 and 1950 and only three in the last four years; improved technique could account for this, but the figures are very good, especially as only one baby was delivered by Caesarean section.

TABLE IV.  
*Causes of Maternal Mortality.*

Pathological Condition.	Number of Cases.
Toxaemia .. .. .	3
Shock and collapse .. .. .	1
Pulmonary thrombosis .. .. .	2
Exhaustion and myocarditis .. .. .	1
Puerperal insanity .. .. .	1
Total .. .. .	8

**Toxaemia of the Mother.**—There were 16 cases of toxæmia; of the babies in those cases, six died *in utero* from seven to eight months (one mother being eclamptic); there were five cases of eclampsia in this group, including the one mentioned. With the great improvement in the treatment of early premature babies, this figure should be greatly lowered by earlier induction of labour.

**Cause Not Known.**—In 17 cases the cause of stillbirth is not known. There is no evidence to account for these cases; it is not lack of notes, but simply that no explanation for the stillbirth could be found. Recent post-mortem results suggest that abnormalities and injury would account for many of these cases.

TABLE V.  
*Stillbirths.*

Cause.	Total, Ten Years.	Three Years, 1945 to 1947.	Three Years, 1952 to 1954.
Prolapse of the cord ..	11	3	3
Toxaemia of the mother ..	16	4	5
Not known .. .. .	17	5	6
Difficult delivery ..	11	2	2
Intrauterine asphyxia ..	1	0	1
Placenta previa .. ..	1	1	0
Accidental hemorrhage ..	1	0	1
Abnormality in the fetus ..	2	0	0
Illness of the mother ..	4	0	2
Cord tightly round neck ..	9	3	2
A fall by the mother ..	1	1	0
Premature rupture of the membranes .. .. .	3	0	0
Total stillbirths ..	77	19	22
Total cases ..	4652	1065	1688

**Difficult Delivery.**—There were 11 cases in which delivery was difficult. This figure seems high. There were four breech deliveries. Analysis of all these cases suggests in retrospect that more vigilant ante-natal observation and early induction of labour would have helped, and in at least one case Caesarean section was indicated. Seven of these occurred in 1948, 1949 and 1950, three in the last-mentioned year; this in 478 cases would be responsible for the rise in the rate per 1000. The Caesarean section rate for this series is only 2.4%, which is significant in this section.

**Illness of the Mother.**—In four cases stillbirth was due to illness of the mother. One occurred at six months

with severe dysentery of the mother; one occurred at seven months, the mother having a high fever; the other two mothers were the same women with a positive Wassermann response. The last-mentioned two stillbirths possibly could have been avoided by more adequate treatment of the mother.

TABLE VI.  
*Neonatal Deaths.*

Cause.	Total, Ten Years.	Three Years, 1945 to 1947.	Three Years, 1952 to 1954.
Prematurity .. .. .	28	12	7
Birth injury .. .. .	17	5	7
Abnormality .. .. .	12	1	6
Hyaline membrane .. .. .	1	0	1
Icterus gravis neonatorum ..	2	1	0
Hydrops fetalis .. .. .	1	0	1
Hæmorrhagic disease .. .. .	2	0	2
Toxaemia in the mother ..	5	2	0
Placenta prævia .. .. .	3	1	0
Atelectasis .. .. .	2	0	1
Total neonatal deaths ..	73	22	25
Total cases ..	4652	1065	1688

**Cord Tightly Round the Neck.**—There were nine cases in which stillbirth was due to the cord being tightly round the neck. This seems to be a very high figure. Foetal distress in the second stage should suggest this complication.

**Other Causes of Stillbirth.**—It is not considered that comment on the other headings would be of value, except to mention the two abnormalities. One fetus was grossly abnormal and macerated; the other was very small and ill-developed with a very small placenta, which did not appear to be able to maintain life any longer.

TABLE VII.  
*Mortality.*

Year.	Neonatal Deaths.	Rate per Thousand Live Births.	Stillbirths.	Rate per Thousand Live and Stillbirths.	Total Deaths.	Total Cases.
1945	8	26.3	5	16.1	13	308
1946	8	23.2	5	14.3	13	345
1947	6	15.0	9	21.7	15	412
1948	4	9.1	9	20.2	13	438
1949	8	17.2	10	21.0	18	471
1950	7	14.9	12	24.8	19	478
1951	7	13.7	5	9.7	12	512
1952	8	14.6	10	18.1	18	548
1953	10	18.9	3	5.6	13	528
1954	7	11.4	9	14.5	16	612
Total	73	15.7	77	16.4	150	4652

#### *Causes of Neonatal Death.*

**Prematurity.**—There were 28 neonatal deaths due to prematurity. This is the greatest cause of neonatal death. Apart from 1946 the annual figure varies from two to four, while the number of patients confined has almost doubled. This shows an improvement in the conduct of these cases. In 1954, when 612 patients were confined, there were no deaths from prematurity alone. The reasons for this improvement are considered to be as follows. (i) Medical practitioners were aware that these figures must be improved, and the nursing staff willingly cooperated in this object. (ii) Oxygen was difficult and at times impossible to obtain in the earlier years; even today it cannot be carried on passenger trains, which seems ridiculous as aircraft will carry it at £7 freight charge. A good deal of industrial oxygen was used, and in retrospect some concern is felt at the possible contamination of these bottles with acetylene. In 1951 the first all-



TABLE VIII.  
Causes of Stillbirths.

Year.	Prolapse of the Cord.	Toxæmia in the Mother.	Not Known.	Difficult Delivery.	Intra-uterine Asphyxia.	Placenta Prævia.	Accidental Hæmorrhage.	Abnormality of the Fœtus.	Illness in the Mother.	Cord Tightly Round Neck.	Fall by the Mother.	Premature Rupture of the Membranes.
1945 .. ..	—	1	—	1	—	1	—	—	—	1	1	—
1946 .. ..	2	—	2	—	—	—	—	—	—	1	—	—
1947 .. ..	1	3	3	1	—	—	—	—	—	1	—	—
1948 .. ..	2	—	3	3	—	—	—	—	—	1	—	—
1949 .. ..	1	2	2	1	—	—	—	1	—	—	—	2
1950 .. ..	2	3	0	3	—	0	—	1	2	1	—	—
1951 .. ..	—	2	1	—	—	—	—	—	—	1	—	1
1952 .. ..	—	3	2	1	—	—	1	—	2	1	—	—
1953 .. ..	2	—	1	—	—	—	—	—	—	1	—	—
1954 .. ..	1	2	3	1	1	—	—	—	—	1	—	—
Total ..	11	16	17	11	1	1	1	2	4	9	1	3

TABLE IX.  
Causes of Neonatal Deaths.

Year.	Prematurity.		Birth Injury.	Abnormality.	Hyaline Membrane.	Icterus Gravis.	Hydrops Fœtalis.	Hæmorrhagic Disease.	Toxæmia in the Mother.	Placenta Prævia.	Atelectasis.
	Number.	Months of Gestation.									
1945 .. ..	4	8, 7, 6, 7	3	1	—	—	—	—	—	—	—
1946 .. ..	5	6, 6, 6, 7½, 7½	1	—	—	—	—	—	2	—	—
1947 .. ..	3	7½, 8, 8	1	—	—	1	—	—	—	1	—
1948 .. ..	3	7, 6½, 6½	—	—	—	—	—	—	—	—	—
1949 .. ..	2	8, 7	3	1	—	—	—	—	—	2	—
1950 .. ..	2	7, 8	1	2	—	—	—	—	2	—	—
1951 .. ..	2	8, 7	1	2	—	—	—	—	1	—	1
1952 .. ..	3	6, 7, 8	3	—	—	—	1	1	—	—	—
1953 .. ..	4	6½, 7½, 6½, 6½	2	2	1	—	—	1	—	—	1
1954 .. ..	0	—	2	4	—	—	—	1	—	—	—
Total ..	28	—	17	12	1	2	1	2	5	3	2

TABLE X.  
Ante-Partum Hæmorrhage Due to Placenta Prævia.

Ante-Partum Hæmorrhage Due to Placenta Prævia.	Primiparae.	Multiparae.	Total.	Stillbirths.		Neonatal Deaths.		Living Babies.
				Primiparae.	Multiparae.	Primiparae.	Multiparae.	
Number of cases ..	8	28	36	—	1	1	3	31
Number of patients treated by Caesarean section ..	6	15	21	—	—	1	2	18

metal oxygen cot was purchased, and in 1952 another was introduced. These cots were an improvement, and in January, 1954, two "Oxygenaire" models were added, and now four early premature babies can be accommodated at once. It is hoped in the near future that a "Humidicrib" will be added. (iii) Better laboratory facilities, a much improved transfusion service and intravenous therapy generally, all give promise of a brighter outlook in the future. Recently in a toxæmic mother labour was induced at seven and a quarter months and the baby survived; another baby, born prematurely at six and a half months and weighing two pounds eight ounces, is now seven weeks old and has gained one pound three ounces in weight. Why so many women here come into premature labour is difficult to explain; a few are toxæmic, but in many cases no cause can be found; in some cases it appears that premature labour is habitual. A possible explanation is that many women in this district work in the fields, or do the cooking and washing for cane-cutters. One patient gave a history of rising at 4 a.m. and finishing at 9.30 p.m., doing all the cooking and washing for nine men.

In the interpretation of the figures for prematurity, if twenty-eight weeks is taken as the standard of viability, it will be seen that 10 infants were not viable, seven

were just barely viable, and eleven were definitely viable. It is believed that this will give a clearer picture of the greatest cause of infant death in this series of cases.

**Birth Injury.**—Death was due to birth injury in 17 cases. As this occurs in small premature babies and in simple, easy confinements, it is difficult to postulate a method of improving this result, except to stress the importance of intelligent anticipation in avoiding difficult deliveries and of episiotomy in premature birth.

**Abnormality.**—Death was due to abnormality in 12 cases. At first glance this section does not offer much hope of improvement; but with the known effects of rubella, the possible effect of the simpler virus diseases in early pregnancy gives food for thought, and an investigation following this line of thought is under consideration.

**Toxæmia of the Mother.**—In five cases death was due to toxæmia of the mother. Increased confidence in the survival of premature babies has justified earlier induction of labour in these cases; also the earlier treatment of these patients has produced improvement of results, as no case has occurred since 1951.

**Other Causes.**—The remainder of the causes of neonatal death are considered to be within reasonable limits. A

number of tables have been included in the desire to give the maximum information in the smallest possible space.

#### Toxæmia.

The mild cases of toxæmia are difficult to collect, and the results would be meaningless without a list of the patients treated at home.

The patients with severe toxæmia here mentioned are those who were admitted to hospital and who required more active treatment than simple sedation and rest.

There were 74 cases of severe toxæmia, not including eclampsia. Of these, 45 were in *primipara* and 29 in *multipara*. There were three maternal deaths, 13 stillborn babies and five neonatal deaths due to these toxæmias.

#### Eclampsia.

There were 13 cases of eclampsia; two of the patients had post-partum fits only. Ten of the patients were *primipara* and three *multipara*; curiously enough, all three *multipara* were pregnant for the fourth time. Ten of the babies survived, and three were stillborn.

Two cases occurred in summer, five in autumn and six in winter.

The influence of toxæmia on maternal mortality is obvious, as five of the eight mothers who died showed a severe grade of toxæmia. The two with eclampsia who died did so of toxæmia, and one in the severe toxæmia group with nephritis also died of toxæmia. Of the remaining two mothers who died, one sustained a pulmonary thrombosis after Cæsarean operation and the other was considered to have died from exhaustion and shock. Both of these patients had a severe grade of toxæmia.

#### Ante-Partum Hæmorrhage.

Ante-partum hæmorrhage occurred in 58 cases, 18 of the patients being *primipara* and 40 *multipara*. Five babies were stillborn (one to a *primipara*, four to *multipara*). There were eight neonatal deaths (four among *primipara*, four among *multipara*). Living babies born numbered 46, including one set of twins. There were no maternal deaths from ante-partum hæmorrhage.

Of the 58 cases of ante-partum hæmorrhage, 36 were due to *placenta previa*; 21 of the patients were treated by Cæsarean section. Details of these cases are shown in Table X.

Of the 22 cases which were not due to *placenta previa*, two were accidental concealed hæmorrhages, six were regarded as being due to toxæmia, and in the remaining 14 cases no real cause was found. Some of these may have been due to *placenta previa* of the marginal type which was not definite enough to be diagnosed. In these 22 cases, there were three stillbirths, two of the infants being premature (six months); five neonatal deaths occurred, three of the babies being born at seven months and one at six months (Table X).

#### Post-Partum Hæmorrhage.

The patients with post-partum hæmorrhage include all those who had a blood loss above normal and who required more than the routine administration of ergometrine and "Pitocin" for their control.

There were 202 cases of post-partum hæmorrhage; of these 68 were in *primipara* and 134 in *multipara*. The most severe case was in a *primipara*, who required 15 pints of blood, four pints of serum and subtotal hysterectomy before the hæmorrhage was controlled and she recovered.

#### Miscellaneous.

##### The Placenta.

The placenta was removed manually from 36 patients; there were no untoward incidents associated with these removals.

##### Hæmorrhage of the Newborn.

There were 11 cases of hæmorrhage of the newborn reported, with two deaths. These hæmorrhages varied

from a few spots of blood in the napkin to a massive sudden *malena* which was almost immediately fatal. The treatment varied from the administration of vitamin K in the minor cases to the administration of vitamin K and the injection of whole blood into the buttock in the more severe cases. In one case blood transfusion was given.

#### Icterus Gravis.

There were 13 cases of *icterus gravis* in this series. One fetus had *hydrops fatalis* and died; the mother was a *primipara*. Two babies with *icterus gravis* died. All patients were treated by exchange transfusion or by a series of simple transfusions of blood, with the exception of one of those with *icterus gravis*, who died in 1948; this child was not given a transfusion.

#### Acknowledgement.

A debt of gratitude is due to the following practitioners for permission to use their cases: Dr. G. A. Bolton, Dr. Ernest Chenoweth, Dr. Ian Chenoweth and Ruth Chenoweth, Dr. R. Courtice, Dr. M. J. Gallagher, Dr. J. V. Guinane, Dr. R. Grant and Dr. Ian Grant, Dr. D. Jacklin, Dr. C. F. Reye, Dr. C. Ruffe and Dr. L. Ruffe, Dr. C. E. Williams and Dr. S. C. Williams, and Dr. K. Whitehead, and to Miss Margaret Hopkins for her assistance in compiling these records, and particularly to the nursing staff of the Mater Misericordiae Private Hospital for very well kept records.

### AN INVESTIGATION INTO THE INCIDENCE OF ECLAMPSIA IN GENERAL PRACTICE IN NEW SOUTH WALES AND QUEENSLAND.

By THE RESEARCH COMMITTEES OF THE NEW SOUTH WALES AND QUEENSLAND FACULTIES OF THE COLLEGE OF GENERAL PRACTITIONERS.

#### Origin of the Investigation.

DURING a week-end course held at Wollongong early in 1955 by the Federation of Country Local Associations, the comment was made by Professor B. T. Mayes that one did not know the incidence of eclampsia in general practice. The death rate due to eclampsia was known through the Committee of Investigation into Maternal Mortality. There is not in New South Wales any method of obtaining the overall incidence of eclampsia, which is not a notifiable disease. The larger teaching hospitals can present the incidence of eclampsia in their institutions, but this includes a number of emergency cases and does not give a truly representative indication of its overall frequency in the general run of obstetric practice. At the meeting of the Federation of Country Local Associations were a number of members of the College of General Practitioners (New South Wales Faculty). One of the activities of the college is the conduct of research in the field of general practice. This is of the clinical or the statistical type, and carried out either individually or collectively. The Research Committee of the newly formed New South Wales Faculty thought that it could be of use in obtaining some figures regarding the incidence of eclampsia, and offered its assistance to Professor Mayes for this purpose. Professor Mayes had previously indicated his willingness to serve on the Advisory Panel of the Research Committee of the New South Wales Faculty, and is himself a member of the college. This project was therefore undertaken with his guidance and cooperation.

#### Scope of the Investigation.

The scope of the investigation was then decided upon. It was thought that the best method of obtaining sufficient satisfactory figures was to send a questionnaire to all the members of the New South Wales Faculty. It was decided to ask members to enlist the cooperation of their colleagues in neighbouring centres by inducing them to fill in returns for their practices also.



The nature of the questionnaire was influenced by several factors. It had to be simple, because it was meant to be attractive to people who did not normally engage in research or statistical surveys, and who might be prejudiced by a complicated form. It was decided to concentrate on obtaining only the following data: (a) the total number of confinements conducted during the period of the survey—namely, the year ended December 31, 1954; (b) the number of cases of eclampsia occurring in the series; (c) whether or not the cases occurred in booked or "emergency" cases; (d) the season of the year in which the cases of eclampsia occurred; (e) the geographical district of the practice concerned.

It was hoped that sufficient figures would be obtained to give a statistically accurate assessment of the incidence of eclampsia in the average general practice.

#### Booked or "Emergency" Cases.

With the almost universal awareness of the dangerous implications of toxæmia of pregnancy, it is probable that the incidence of eclampsia has decreased greatly in the past twenty-five years (Mayes, 1950) as the result of adequate treatment of the preeclamptic patient. We were therefore interested to discover whether lack of cooperation by patients in ante-natal care, or lack of facilities for adequate ante-natal care, was a factor in the occurrence of frank eclampsia.

#### Season of the Year.

There has long been a clinical impression that eclampsia occurs most frequently in winter. Therefore, the question of season was included, although we hoped that there would be scarcely enough eclamptics to give a statistically significant assessment of the seasonal incidence.

#### Geographical District.

It was thought that information as to the geographical district was necessary to find whether the results were truly representative of the whole State. It might also give information as to whether inadequate hospital facilities were a contributing cause of eclampsia. Possibly climatic factors might also have influenced the incidence.

#### Anonymity of Returns.

It was decided not to ask participants to reveal their identity. It was thought that practitioners might be reluctant to reveal cases of eclampsia, either through a sense of reflection on their capabilities, or through an inherent mistrust in anything approaching "authorities". It was known that survey forms were being sent only to reliable practitioners, or by them to chosen colleagues. The possibility of forgery or foolery appeared unlikely. Actually a number of participants did sign their returns, and added useful comments, to which reference will be made later.

#### The Queensland Faculty.

The New South Wales Faculty keeps in touch with its sister faculty in Queensland. The Chairman of the Research Committee of the Queensland Faculty, Dr. Paul Hopkins, was interested to hear of our survey and had recently published a review of obstetrics in general practice (Hopkins, 1955). It was agreed that the Queensland Faculty should carry out a similar survey, and that we should combine our results in one publication.

Such is the origin and scope of this survey.

#### New South Wales Results.

##### Distribution of Results.

The total number of returns, confinements and districts of practice is shown in Table I. It will be seen that 88 returns accounted for 5772 confinements. It is not possible to state how many doctors are concerned, because in some instances a return covered a partnership of two or three doctors. One return (from Inverell) covered the total confinements at the Inverell District Hospital, and represents the work of 10 doctors not separately reported. In this case the figure of 10 is included in the total returns as

10 returns. In all other cases the return from each practice has been included as one return, irrespective of the number of doctors in the practice.

The district of practice shows that the greatest number of returns comes from the central west and the northern and central tablelands. This loading is due to the enthusiasm of some members in coopting the help of their colleagues. The result is that the subsequent figures do not give any reliable indication of the geographical incidence of eclampsia.

TABLE I.

New South Wales.

Total number of Returns, 88; total number of Confinements, 5772.

District.	North.	Central.	South.
<i>Country:</i>			
Coast .. .. .	5	3	—
Tablelands .. ..	13	16	3
West .. .. .	2	31	3
<i>Other:</i>			
Metropolitan .. 10			
South Australia .. 1			
Unspecified .. 1			

#### Incidence of Eclampsia.

The number of eclamptics and their distribution by district and season are shown in the following tabulation:

Total number of confirmed eclamptics .. . . .	6
Number of eclamptics among unbooked patients ..	1
Distribution of cases of eclampsia according to district:	
North Coast .. . . .	2
Northern Tablelands .. . . .	1
Central West .. . . .	3
Distribution of cases of eclampsia according to season:	
Summer .. . . .	5
Winter .. . . .	1
Autumn and spring .. . . .	0

In the counting of the cases of eclampsia, two cases notified have been deliberately excluded. In one a footnote on the return commented that the urine was free of albumin and the blood pressure was 120 millimetres of mercury, systolic, and 80 millimetres, diastolic, the patient subsequently suffering from puerperal insanity, from which she recovered. In the other, the comment was made that the patient had a "turn" while recovering from the anaesthetic, that only a nurse was present and that the blood pressure was normal. It is thought that other diagnoses than eclampsia are possible, and these cases have therefore not been included.

The six cases correspond to an incidence of one in 962, or 1.04 per 1000 confinements. It is thought that this is a truly representative figure of the incidence of eclampsia in general practice in New South Wales.

#### Further Features of the Cases of Eclampsia.

As a matter of interest the geographical distribution of the cases of eclampsia was as follows: north coast, two; northern tablelands, one; central west, three. This, of course, gives no indication of the geographical incidence of eclampsia for the reasons already mentioned. With regard to the season, it was found that five of the cases occurred in summer and one in winter. It is doubtful whether this is of statistical significance in so few cases; but it does suggest that eclampsia is not of predominantly winter occurrence in New South Wales.

One patient only was unbooked. This patient was first examined when in labour with twins, and "all recovered" (presumably including the medical attendant). Another patient, although booked, failed to attend owing to the absence of the principal on holiday, and presented to the *locum tenens* with preeclamptic signs. This practitioner,

who averages 100 midwifery cases *per annum*, also notes that his previous case of eclampsia had occurred ten years previously in an unbooked case. This again corresponds to the apparent overall incidence of 1.04 per 1000 confinements.

#### Queensland Results.

The Queensland results are shown in Table II.

#### Distribution.

The number of confinements from the metropolitan area (108) is too small to be of value. In the country areas the numbers (4245) are better. A wide geographical distribution has been obtained. Again in some places the numbers are small, but in many of these areas the population of the towns is less than 2000.

Geographically, seven cases of eclampsia occurred in the north and central coastal areas, one in the northern tablelands and one in the southern tablelands. As there were 3070 cases involved in coastal areas, perhaps these figures have some significance.

TABLE IIA.  
Queensland.

Area.	Number of Confinements Reported.	Number of Cases of Eclampsia.
Metropolitan (Brisbane) ..	108	0
Country .. .. .	4245	9
Total .. .. .	4353	9

#### Season.

Six cases occurred in winter, one occurred in summer and two were unspecified.

#### Booked or Unbooked.

Seven eclamptics were booked patients. In two instances this information is not available. However, even booked patients in isolated areas may be unable to attend for ante-natal examinations as often as is desirable.

#### Other Features.

Some other features of eclampsia in Queensland may be of interest. Dr. J. H. Steel, of Winton (central tablelands), told us that in three years he had not seen one patient with severe toxæmia, and only a few with mild toxæmia.

In Cloncurry (far north-west) Dr. D. Harvey Sutton has confined approximately 500 patients in six years, with two cases of eclampsia. One of these patients was an aboriginal woman, unbooked, from Birdsville (far south-west—Flying Doctor service, no doubt); the other was a booked local white woman with twins. Neither of these two cases occurred in 1954.

A ten years' series from a private hospital in Mackay (north coast), from January, 1945, to December, 1954, covers 4652 confinements (Hopkins, 1956). There were 13 cases of eclampsia during this ten-year period. Two of these occurred in summer, five in the autumn and six in winter. Ten of these patients were *primiparæ* and three *multiparæ*.

It is realized that it is unsound to base conclusions on a small number of cases, but it does seem reasonable to suggest that rigid ante-natal supervision is essential in the north and central coastal areas, especially in the autumn and winter months.

#### Overall Incidence.

The overall incidence of eclampsia was one in 484 confinements, or 2.07 per 1000.

#### Combined Results for New South Wales and Queensland.

#### Incidence.

The total number of confinements recorded for the two States, New South Wales and Queensland, is 10,125, and the total number of eclamptics is 15. This corresponds to an incidence of one in 675 or 1.48 per 1000 cases.

#### Booked or Unbooked.

Most of the Queensland cases included in the series, and five of the six New South Wales cases, were in booked patients. However, the comments and the additional cases mentioned suggest that unbooked cases and multiple pregnancies are still potential sources of eclampsia, and merit further propaganda to avoid unbooked cases and to promote additional ante-natal care of patients with multiple pregnancies. Furthermore, in North Queensland, distance from medical care is probably a factor. Ante-natal visits may be sporadic even in booked cases, and a long journey has to be made to hospital when the time is ripe.

#### Geographical Distribution.

The geographical distributions have not been combined, because the New South Wales figures are not representative of the amount of obstetrics carried out in each area.

TABLE IIB.  
Queensland: Distribution of Country Cases.

	North.			Central.			South.		
	Town.	Cases.	Eclampsia.	Town.	Cases.	Eclampsia.	Town.	Cases.	Eclampsia.
Coast .. ..	Mackay (18,500) <sup>1</sup> ..	866	1 (W) <sup>a</sup>	Rockhampton (40,676)	280	2 (W)	Cooroy (1069) ..	36	0
	Mossman (1461) ..	86	1 (U) <sup>a</sup>						
	Townsville (40,560)	900	2 (W) <sup>a</sup>	Gayndah (1644)	83	1 (W)	Gympie (9964)	70	0
			(S) <sup>a</sup>						
	Cairns (22,000) ..	740	0						
Tablelands ..	Cooktown (397) ..	9	0						
	Hughenden (1772)	81	0	Clermont (1587) ..	92	0	Oakey (1640)	48	0
	Atherton (2527) ..	75	1 (U)	Winton (1396) ..	54	0	Darling Downs (town unspecified)	73	0
	Marreeba (3367) ..	174	0				Toowoomba (48,152)	164	1 (W)
	Herberton (900) ..	83	0						
West .. ..	Julia Creek (whole Shire) (1645)	36	0	Blackall (1885) ..	109	0	Cunnamulla (1955)	104	0
	Cloncurry (1955) ..	82	0						
Total .. ..	—	3132	5	—	618	3	—	495	1

<sup>1</sup> Figures in parentheses denote population.

<sup>a</sup> W: Winter; S, Summer; U, Season Unspecified.



The Queensland figures suggested a higher incidence in the central and northern coastal areas of that State. This may have been due to a climatic factor.

#### Seasonal Incidence.

It has generally been accepted that eclampsia has a cold weather incidence. Professor Mayes informs us that this has been verified in Sydney and Capetown, but that Dieckmann states that it is likely to be associated with a change in meteorological conditions rather than with any given temperature or humidity (Mayes, 1955). The high incidence in North Queensland would suggest that humidity is a factor. Our figures are too small to allow any dogmatic conclusions to be drawn. This point would have to be investigated in a far larger series of eclampsies to be of any statistical significance.

#### Comparison with New Zealand.

It is of interest to compare our figures with those of our sister dominion of New Zealand. In that country, eclampsia has for many years been a notifiable disease. Figures are available up to 1951 in the Medical Research Council report on "Eclampsia in New Zealand 1950-1951", by Corkill (1952). The incidence of eclampsia in New Zealand is given in that publication as follows:

1928 to 1939	.. .. .	3.07 per 1000
1940 to 1947	.. .. .	1.12 per 1000
1950 to 1951	.. .. .	1.40 per 1000

We are indebted to Professor Mayes for information received in a personal communication from Dr. Corkill relating to the 1952-1954 figures, which show an incidence of 1.43 per 1000.

#### Summary.

An attempt has been made to determine the incidence of eclampsia in general practice in New South Wales and Queensland.

The method used was to send circulars to all members of the New South Wales and Queensland Faculties of the College of General Practitioners.

Information has been received regarding 10,125 confinements, 5772 in New South Wales and 4353 in Queensland, conducted during the year 1954.

The incidence of eclampsia for those series was 1.04 per 1000 in New South Wales and 2.07 per 1000 in Queensland. The incidence in the combined series was 1.48 per 1000.

Information regarding the geographical and seasonal incidence and regarding the relative frequency of eclampsia in booked and unbooked cases was also obtained. The number of eclampsies was too few to allow any firm conclusions to be drawn regarding these contributing factors. However, certain possible aspects of their significance have been put forward.

#### Acknowledgements.

The Research Committees of the New South Wales and Queensland Faculties of the College of General Practitioners extend their thanks to the many members and their colleagues who have contributed to the compilation of this report, and to Professor B. T. Mayes and the Department of Obstetrics, University of Sydney, for their advice in its preparation.

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### IMMUNIZATION OF CHILDREN AGAINST WHOOPING-COUGH WITH PERTUSSIS HÆMAGGLUTININ (H.A.P.A.): FIELD RESULTS OVER THREE YEARS.<sup>1</sup>

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H.A.P.A. is a suspension of floccules of aluminium phosphate on which are adsorbed components of freshly grown virulent *Hæmophilus pertussis* obtained by extracting bacterial bodies with molar, slightly alkaline sodium chloride solution. The immunizing course for children consists of two subcutaneous injections of 0.25 millilitre each, spaced a month apart. Previous papers contain the details of the development, properties and standardization of H.A.P.A. and its effects in children and laboratory animals (Keogh and North, 1948; Fisher, 1950; Warburton and Fisher, 1951; Fisher, Warburton, Wettenhall and Williams, 1951). Early results suggested that H.A.P.A. would be an efficient prophylactic in children against whooping-cough (Fisher, 1952).

Over the past three years H.A.P.A. has been issued to a number of medical practitioners in different States for use in their practices, and also to five municipal councils in Victoria for use in immunization campaigns.

#### Results of the Survey.

The present survey was conducted along two independent lines. In the first, all private doctors who had received a total of five millilitres or more of H.A.P.A. were sent circulars and their opinions were sought. They were asked specifically whether they had encountered in their practices children, unimmunized or immunized with H.A.P.A., who had been exposed to contact with clinical cases of whooping-cough in their homes and, if so, whether the children had contracted the disease. This inquiry was based on recent findings which showed that, in home exposures, the majority of unprotected children developed clinical whooping-cough, the attack rates being 87.3% in 173 home exposures (Medical Research Council, 1951), 78.6% in 112 home exposures (Kesson and Harling, 1953), and 94.7% in 57 known contacts (Bousfield and Holt, 1954).

Of 66 questionnaires sent out, 48 were returned. The general opinion on H.A.P.A. was uniformly favourable. In 13 of the replies 35 home contacts were reported, with the results shown in Table I.

Included in the foregoing figures are those relating to two doctors' families. One child in each who had been immunized with H.A.P.A. did not contract pertussis from a sibling suffering from the disease, but another child in each family, who had not been immunized with H.A.P.A., did contract the disease.

The incidence of whooping-cough in the unimmunized children was 87%, and in the H.A.P.A. immunized group 10.5%. The difference in incidence between the two groups is statistically highly significant ( $P < 0.001$ ). In spite of the small numbers observed, we regard this result as a strong indication that H.A.P.A. is a good prophylactic. There appears to have been no obvious source of bias to influence this result. The availability of H.A.P.A. was not generally known, and was brought to the notice of the doctors who used it mainly by personal contact with those responsible for its early development. We do not think that this factor would load the result in any way.

In the other part of the survey the protective efficiency of H.A.P.A. was investigated in the five municipalities.

<sup>1</sup> Read at a meeting of the Section of Public Health and Industrial Medicine, Australasian Medical Congress (British Medical Association), Ninth Session, Sydney, August 20 to 27, 1955.

using it in council immunization campaigns. At the time of the immunization the age of the majority of the children was under twelve months, and practically all the children were aged under two years. Cases of pertussis came to notice mainly through notification, but unnotified cases detected in other ways were also included. Children aged under five years were taken as the group at risk. Accurate figures for the numbers of children in this age group were available for 1954, from the Commonwealth census taken in that year, and were given to us by Mr. R. E. Lucchinelli, of the Commonwealth Statistician's Office in Melbourne. The numbers of children at risk in the periods before and after 1954 were estimated from the

TABLE I.

Subjects.	Total Number of Home Contacts.	Number of Children who Developed Pertussis.
Children immunized with H.A.P.A.	19	2
Unimmunized children	16	14
Total	35	16

1954 census figures, by means of trends in the number of school entrants, supplied to us by Mr. W. Phillips, of the Department of Education of Victoria. For each year the cumulative number of children fully immunized with H.A.P.A. was deducted from the number at risk in the municipalities. The cases of pertussis were then checked against council immunization records and assigned either to the "immunized" group if they had received the full course of H.A.P.A., or to the "other" group if they had not.

The age of the oldest child who had received H.A.P.A. and subsequently developed whooping-cough was eight years. Therefore, it seemed desirable to conduct the survey on children aged up to nine years; however, no accurate figures were available for the number of children in that age group. The incidence of whooping-cough in the nought to five years and nought to nine years age groups was as shown in Table II.

TABLE II.  
Number of Cases of Pertussis.

Subjects.	0 to 5 Years Age Group.	0 to 9 Years Age Group.	Difference (Percentage).
Children fully immunized with H.A.P.A.	9	12	25.0
Others	204	277	26.3

Since the percentage fall in the number of cases from the higher to the lower age group was practically the same in the H.A.P.A. immunized children and the others, no significant error was introduced by carrying out the survey on the lower age group.

The results in four municipalities are shown in Table III. In the fifth area (Malvern) there were insufficient cases to warrant its inclusion in the survey. Fern Tree Gully is a near-metropolitan country district; the other three are metropolitan, largely industrial in character. The attack rates are expressed as numbers of cases per 1000 child-years. In one municipality (Brunswick) the incidence of whooping-cough was slightly higher in the H.A.P.A. immunized group than in the other. In the other areas the ratio of incidence of pertussis in the H.A.P.A. immunized groups to the other varied from 1:1.98 to 1:6.48. On all cases the ratio was 1:3.7. In all, nine cases occurred in 4920 surveyed child-years among the H.A.P.A. immunized, and 204 cases in 30,063 child-years among the others.

When these figures are considered, certain circumstances must be taken into account, the more important of which are as follows.

First, a number of children not fully immunized with H.A.P.A. may have been immunized with other whooping-cough prophylactics, or may have received one injection of H.A.P.A. Second, only children fully immunized at the beginning of the periods referred to in Table III were included in the "immunized" group; those immunized during these periods were placed in the "other" group. Third, we have reason to believe that in one of the areas the number of children fully immunized with H.A.P.A. was understated. Fourth, many more cases of whooping-cough probably occurred than were notified; we believe that cases occurring in immunized children were more likely to be notified than cases occurring among the non-immunized. Fifth, the exposure rates in the group fully immunized with H.A.P.A. and in the other group may not have been identical; general hygiene and the rate of immunization of siblings may have been lower in the homes of children not immunized with H.A.P.A. The first three of these factors would certainly, and the fourth possibly, show H.A.P.A. in an unduly unfavourable light as a prophylactic; the fifth probably in an unduly favourable light. On the whole we consider it unlikely that the figures should be biased in favour of H.A.P.A.

#### Discussion.

It was not possible to submit H.A.P.A. to a fully controlled field trial. However, our home exposure survey indicated that it conferred a significant and satisfactory degree of immunity on children against whooping-cough. This conclusion was supported by the performance of H.A.P.A. in the council immunization campaigns. Under the circumstances, the evidence gained in the latter must be considered less reliable than the figures obtained from the former. No information is available with regard to the relative potency of H.A.P.A. and bacterial vaccines; it may be pointed out, however, that only two injections of H.A.P.A. were given to provide the protection demonstrated in the children, whereas it is usual to administer three or four injections of bacterial pertussis vaccines.

One further point merits discussion. Three experienced overseas observers found independently that H.A.P.A. performed poorly in the mouse assay, with the use of the intracerebral method of challenge (Kendrick, Eldering, Dixon and Misner, 1947; Kendrick, Updyke and Eldering, 1949), which is now widely employed for estimating the potency of pertussis vaccine. Eldering (1952) found that 0.1 millilitre or more of H.A.P.A. was required to protect 50% of the animals against death, and much higher figures were obtained by Standfast (1952) and Ungar (1952). Of bacterial vaccines, doses containing approximately 500 million organisms appear to be required to produce the same effect (Pittman, 1954). Therefore, in the case of H.A.P.A. the mouse protective dose is at least 20% of the full human course (0.5 millilitre); the mouse protective dose of a bacterial vaccine is about 1% of the human immunizing course (60 thousand million organisms). If it is accepted that H.A.P.A. was shown to be an effective prophylactic in children, it must be concluded that the intracerebral mouse assay failed to indicate that this would be the case.

#### Summary.

The results of immunization of children against whooping-cough with H.A.P.A. (pertussis haemagglutinin, aluminium phosphate adsorbed) over the past three years have been surveyed.

In one investigation reports were obtained from practitioners concerning home contacts of children with others suffering from clinical pertussis. Of 19 children immunized with H.A.P.A. and so exposed, two contracted pertussis; of 16 not immunized, 14 did so.

In the second investigation, covering four Victorian municipal districts, where H.A.P.A. was used in council immunization campaigns, nine cases of pertussis were found in 4920 child-years in children fully immunized with



TABLE III.  
Results of Immunization with H.A.P.A. in Four Victorian Municipalities.

Municipality.	Period.	Children Fully Immunized with H.A.P.A. <sup>1</sup>				Others. <sup>2</sup>			
		Number at Risk.	Number of Child Years.	Number of Cases of Pertussis.	Number of Cases per 1000 Child Years.	Number at Risk.	Number of Child Years.	Number of Cases of Pertussis.	Number of Cases per 1000 Child Years.
Brunswick .. ..	1954 1955 (to mid-March)	205 500	205 } 305 100 }	1 } 2 1 }	6.56	3915 3620	3915 } 4639 724 }	11 } 20 9 }	4.31
Fern Tree Gully ..	1954 1955 (to mid-May)	507 983	507 } 876 369 }	0 } 1 0 }	1.14	2617 2297	2617 } 3478 861 }	2 } 11 9 }	3.16
Fitzroy .. ..	1953 1954 1955 (to mid-May)	130 296 393	130 } 573 296 } 147 }	0 } 2 1 }	3.49	2166 2000 1903	2166 } 4914 2000 } 748 }	4 } 34 6 }	6.92
City of Melbourne..	1952 (from July) 1953 1954 1955 (to end of June)	299 1051 1251 1431	149 } 3166 1051 } 1251 } 715 }	0 } 4 0 } 0 }	1.26	6434 5682 5482 5302	3217 } 17,032 5682 } 5482 } 2651 }	4 } 139 16 } 74 }	8.16
All Municipalities	.. ..	.. ..	4920	9	1.83	—	30,063	204	6.78

<sup>1</sup> At the beginning of the period referred to.

<sup>2</sup> Include children not immunized against whooping-cough; children immunized against whooping-cough with agents other than H.A.P.A.; children immunized against whooping-cough with only one injection of H.A.P.A.

H.A.P.A., and 204 cases in 30,063 child-years in other children.

In the light of these results H.A.P.A. was considered an efficient prophylactic in children. The fact that H.A.P.A. had been found to perform poorly in the laboratory assay involving the intracerebral mouse test was discussed.

#### Acknowledgements.

Space does not allow us to thank individually all the doctors, nurses and public health and other officials who supplied us with information and help. We are particularly grateful for their cooperation, without which this survey could not have been carried out. We also wish to thank Dr. E. V. Keogh, Dr. S. W. Williams and Dr. R. J. Farnbach for their continued interest and advice.

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#### THE PRACTICAL MANAGEMENT OF INTRAVENOUS FLUID THERAPY IN INFANCY AND CHILDHOOD.

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THIS paper describes, in outline, the practical management of intravenous fluid therapy as practised at the Royal Alexandra Hospital for Children, Sydney. The intention is to make this approach as simple as possible, and as a result it is likely that certain inaccuracies will be introduced; such inaccuracies are part of the price which must be paid in any attempt to simplify a complicated problem.

Only two solutions are now used in routine work; these are half-strength physiological saline with 2.5% glucose (loosely called "N/2, saline with glucose") and quarter-strength physiological saline with 3.75% glucose (loosely called "N/4, saline with glucose"). Potassium solutions are available in ampoules; the calculation of the amount of potassium to be given is very simple, and this calculated amount can then be transferred by means of a syringe to the flasks of "N/2, saline with glucose" or "N/4, saline with glucose".

I should like to stress the fact that subcutaneous infusions have not been satisfactory in my experience; at this hospital the subcutaneous route is now used only if it is not possible, for technical reasons, to administer fluids intravenously. The sites where veins can usually be found in infants and small children are well described in "Handbook of Paediatric Medical Emergencies" by de Sanctis and Varga; the following are the common sites: (i) the long saphenous vein just above and anterior to the tip of the medial condyle of the ankle; (ii) on the dorsum of the wrist, just a little medial to the mid-line; (iii) over the radial styloid process; (iv) at the junction of middle and distal thirds of the radius, just posterior to the coronal plane of the limb.

#### Principles.

The immediate aim of treatment is to supply fluid and appropriate electrolytes rapidly and in adequate quantities in order to rehydrate the patient, and then to continue this administration at such a rate as to meet his normal needs and also to replace pathological losses which are

occurring—that is, loss as fluid stools in gastro-enteritis, or as gastric aspirate in intestinal obstruction.

The three major aspects of treatment are as follows: (i) Rehydration: the aim is to achieve this within three hours of the commencement of treatment, but sometimes a longer period is necessary. (ii) Replacement of pathological losses as they are occurring: this involves careful recording of such losses on a "fluid balance chart", and appraisal of the clinical state of the patient every six hours. (iii) Maintenance needs: a guide to this volume is given in Table II.

#### Recognition of Dehydration and Electrolyte Depletion.

In the clinical assessment of the patient, no single sign is sufficient; all available signs are observed and recorded, and the final appraisal is made after all factors have been considered. The following observations seem important: (i) the mental state—particularly restlessness or apathy; (ii) the degree of thirst; (iii) the state of dryness of the tongue and oral mucosa; (iv) the fontanelle—level and tension; (v) eyeball tension; (vi) skin tone; (vii) the peripheral circulation—temperature, and colour of the skin; (viii) the pulse rate; (ix) the abdominal girth—in a patient suffering from gastro-enteritis, a distended, tympanitic abdomen associated with the frequent passage of fluid stools strongly suggests paralytic ileus due to severe electrolyte disturbance; (x) urinary output; (xi) weight loss.

When the skin is inelastic, the fontanelle is much depressed, the mucous membranes are dry and the patient is lethargic, gross dehydration is present; but significant degrees of dehydration may be found when the only signs are diminished eyeball tension, diminished skin tone, and a little restlessness.

#### Fluids in Common Use for Intravenous Therapy.

In Table I are shown the fluids most commonly used for intravenous therapy.

TABLE I.

Name on Label.	Volume in Flask or Volume. (Millilitres.)	Sodium Concentration. (Milliequivalents per Litre.)	Potassium Concentration. (Milliequivalents per Litre.)	Chloride Concentration. (Milliequivalents per Litre.)
Normal saline <sup>1</sup>	500	154	—	154
Half-strength normal saline with 2.5% glucose <sup>1</sup>	500	77	—	77
Quarter strength normal saline with 3.75% glucose <sup>1</sup>	500	38.5	—	38.5
5% glucose in distilled water <sup>1</sup>	500	—	—	—
Potassium chloride (KCl) <sup>2</sup>	10	—	1	1
Potassium monohydrogen phosphate (K,HPO <sub>4</sub> ) <sup>2</sup>	10	—	1	—

<sup>1</sup> Flasks.

<sup>2</sup> Ampoules.

These solutions are supplied by Farmer Hill and Company, the first four solutions as 500 millilitre rubber-capped flasks and the last two as glass ampoules containing 10 millilitres. The concentrations in the ampoules of potassium solutions are so arranged that one millilitre of such solution supplies one milliequivalent of potassium.

#### Practical Application.

##### Rehydration.

Rehydration is carried out in accordance with the following considerations.

**Volume of Fluid.**—My experience has been that when a child is dehydrated and requires parenteral fluid therapy, the volume of fluid required to rehydrate the child is equal to at least 5% of its body weight. When dehydration is very severe, this volume rises to (and may occasionally exceed) 15% of the child's body weight. To estimate the

volume of fluid needed to rehydrate the patient, it is sufficiently accurate to base calculations on the child's actual weight, determined by weighing him just before commencing intravenous fluid therapy. If the child is assessed as being "a%" below his usual weight because of dehydration, then:

Volume of fluid to be given (in millilitres)

$$= \frac{a}{100} \times \text{weight of child in kilograms} \times 1000$$

$$= a \times \text{weight of child in kilograms} \times 10.$$

It must now be emphasized that this calculated volume can only be a guide to the volume of fluid actually required.

**Nature of Fluid.**—The most satisfactory fluid for this rehydration phase is "N/, saline with glucose"; this is used as a routine at the Royal Alexandra Hospital for Children unless laboratory investigations suggest that some other fluid is more suitable. The concentration of sodium in this solution is such that children who have lost more sodium than water in becoming dehydrated will receive adequate sodium to make good the deficiency; and, on the other hand, the sodium concentration is such that children who have lost more water than sodium are able to excrete any unwanted excess of sodium, provided that their kidneys are functioning normally.

**Rate of Administration.**—An attempt is made to administer this calculated volume of fluid during the first three hours of treatment. This can be achieved by adjusting the rate of the "drip"; a rate of 30 drops per minute will deliver approximately 100 millilitres per hour.

**Observation during Rehydration Phase.**—The patient's clinical condition and state of hydration are reviewed at least every hour during the rehydration phase, and also when the calculated volume has been given. One of the most important observations during this period is the output of urine; a test tube is strapped to the penis of the male baby, and the bladder of the female baby is catheterized, to determine whether urine formation is adequate.

As a result of frequent reassessment it may become clear that rehydration has been achieved before the whole of the calculated volume has been administered; if this happens, the remainder of the volume is, of course, not given. But it often happens that even when the whole of the calculated volume has been given, there is still clinical evidence of dehydration; this means that the initial assessment of dehydration was too low. A further allowance is then calculated on the same principles as for the initial estimate, and this second volume is administered at the same rate as the first volume; hourly observation is continued during this time.

#### Maintenance Requirements.

**Volume of Fluid Indicated.**—In Table II are shown the fluid maintenance needs at increasing ages from birth to twelve years.

**Type of Fluid.**—The fluid in routine use is "N/, saline with glucose", except for infants aged three months or less; such infants cannot tolerate such a high concentration of sodium, and for them it is recommended that half the maintenance volume be given as "N/, saline with glucose", and the other half as 5% glucose in distilled water.

**Rate of Administration.**—The volume of fluid for maintenance needs, and the volume allowed for replacement of continuing losses, are added together and administered over a twenty-four-hour period (see next section).

#### Replacement of Pathological Losses.

**Volume of Fluid.**—Pathological losses are best replaced while they are occurring; if losses are not replaced until the end of a twelve or twenty-four hour period, the child will become dehydrated. To achieve replacement of losses while they are occurring, it is essential to review the patient and the fluid balance chart at least every six hours. When losses are occurring as a result of gastric



or ileostomy suction, mathematical replacement is possible; but when the main loss is from diarrhoea, and fluid stools are being absorbed into napkins, then the volume to be replaced can be at best an intelligent guess. As a guide, I suggest that a child aged twelve months, with moderately severe diarrhoea, may lose 300 millilitres per day; if the diarrhoea is very severe, 1000 millilitres can be lost.

**Type of Fluid.**—When laboratory analysis of gastric and ileostomy fluids is not available, then "N/2 saline with glucose" is a satisfactory fluid. This fluid is also useful to replace loss from diarrhoea.

TABLE II.

Age.	Volume of Fluid for Maintenance Needs of "Average" Children. (Millilitres per Twenty-four Hours.)
Birth to two weeks	Body weight in kilograms $\times$ age of baby in days $\times$ 10.
Two weeks to six months	Body weight in kilograms $\times$ 140 (up to a maximum of 1000 millilitres).
Six months to twelve months	1000 to 1100 millilitres.
Twelve months	1100
Two years	1200
Four years	1400
Eight years	1800
Twelve years	2200

**Rate of Administration.**—As I have already indicated, the volume of fluid estimated to replace pathological losses as they occur is added to the volume estimated to satisfy maintenance; this total volume is then administered over a twenty-four-hour period, after rehydration is achieved. This rate can be calculated as the number of drops per minute; 12 drops per minute will deliver approximately one litre in twenty-four hours.

The fluid balance chart and the clinical condition of the patient are reviewed every six hours; if losses are greater or less than anticipated, it may well be necessary to modify the programme.

#### Potassium.

**Time to Administer.**—Potassium is never administered until it has been demonstrated that the patient is secreting an adequate volume of urine; consequently potassium is seldom given in the rehydration phase. When it is considered that urine is being formed in adequate quantities, potassium is administered to all patients receiving fluid parenterally; in this way past or continuing losses of potassium will be replaced over a period of several days by retention of some of the potassium so given. It is thought that this approach is better than awaiting the development of gross potassium depletion before attempting replacement.

**Amount Given.**—Approximately three millilitres of potassium solution (that is, three milliequivalents of  $K^+$ ) per kilogram of body weight per twenty-four hours are adequate for most patients. Because of the ceiling imposed in the paragraph on concentration of potassium (see below), many of the larger children do not receive as much potassium as three milliequivalents per kilogram; they will receive adequate potassium if this ceiling is observed, even though it may be less than three milliequivalents per kilogram of body weight.

**Choice of Solution.**—Most often equal volumes of potassium chloride and di-potassium monohydrogen phosphate solutions are used; in intestinal and pyloric obstructions, however, chloride losses are so high that it is frequently advisable to give nearly all the potassium as potassium chloride solution.

**Concentration of Potassium.**—Except when there is clinical and/or laboratory evidence of gross potassium depletion, it is usual not to add more than 15 millilitres (that is, 15 milliequivalents of potassium) of potassium solution to a 500 millilitre flask of fluid. The potassium solution

is added by syringe to the flasks of fluid being given for maintenance needs and to replace continuing pathological losses; it is thus given evenly over the whole twenty-four-hour period.

#### Albumin.

Human albumin is now supplied as a 25% solution in rubber-capped bottles containing 100 millilitres and in glass ampoules containing 25 millilitres. The present practice at this hospital is to administer one gramme of albumin (four millilitres of solution) per kilogram of body weight per twenty-four hours; sometimes an amount up to two grammes per kilogram is given. This calculated allowance is injected by syringe into a flask of fluid being given to supply maintenance needs.

#### Other Considerations.

For the sake of simplicity, alterations in acid-base balance, the administration of special solutions such as sodium bicarbonate, sodium lactate and ammonium chloride, the increased fluid allowance necessitated by fever and polyuria, the sodium content of human albumin solution, and the vitamin requirements of patients receiving intravenous fluid therapy, have been omitted. The important problems of the prevention and management of burn shock deserve separate consideration.

#### Summary.

1. The clinical appraisal of dehydration is described.
2. Tables are submitted listing a few simple fluids commonly used, and outlining the requirements of average infants and children for maintenance.
3. The practical management of parenteral fluid therapy is discussed under the headings of rehydration, maintenance needs, replacement of continuing pathological losses, potassium requirement and albumin administration.
4. Great stress is laid on the necessity for frequent review of the clinical state of the patient during intravenous administration of fluids.

#### IMMUNIZATION BY THE BLOOD ANTIGEN KELL IN HÆMOLYTIC DISEASE OF THE NEWBORN AND IN BLOOD TRANSFUSION.

By R. T. SIMMONS.

From the Commonwealth Serum Laboratories, Melbourne.

COOMBS, Mourant and Race (1946) discovered the Kell (K) blood factor, and the corresponding antibody, which was of the incomplete or blocking type, was found in the serum of a mother whose baby was thought to be suffering from hæmolytic disease of the newborn. Wiener and Sonn-Gordon (1947) independently described a "new" blood group called Si, found during the investigation of a blood transfusion reaction. Genetic studies indicated the inheritance of the blood factor as a simple Mendelian dominant, and its frequency (12.9%) in 148 individuals in New York was established. It was subsequently found that the factors K and Si were identical. Race and Sanger (1950) tabulated 17 cases of isosensitization due to the Kell factor, and these had been reported by a number of investigators over a period of about four years. Race and Sanger (1954) state that the K antigen is independent of the antigens relating to the A-B-O, M-N-S, Rh, P, Lutheran, Lewis, Duffy and Kidd blood group systems, and that no evidence for linkage has been found between the Kk genes and the genes for the eight other blood group systems, for sex and for the ability to taste phenylthiocarbamide.

Grundorfer (1955) reported a case of Kell isosensitization, and presented a bibliography covering some 32 examples in which the Kell antibody had been identified. Three of the cases presented in the series had been iden-

tified by Simmons and Jakobowicz (unpublished), and were referred to by Wiener, Brancato and Waltman (1953). The cases producing anti-K were classified as follows: 10 cases of hæmolytic disease of the newborn caused by maternal immunization to the Kell factor; 11 transfusion reactions chiefly due to sensitization to the same factor; nine examples in which the cause of immunization was not stated; two instances in which anti-K was produced by deliberate immunization in volunteers. In a number of instances anti-K was found together with one or more other atypical antibodies.

In an earlier paper, van Loghem, de Raad and van Hattum (1953) referred to other instances of anti-K, and to eight examples identified in Holland during recent years. In six cases anti-K was found together with Rh antibodies, and in two anti-K was the only immune antibody present. In five cases anti-K was produced as the result of blood transfusion, and in three cases anti-K was formed during pregnancy. In 538 individuals tested for the K factor in the Netherlands, 8.5% were shown to be Kell positive.

Grove-Rasmussen, Dreisler and Shaw (1954) found ten examples of anti-K in about 10,000 blood samples tested during a period of fifteen months. In two instances anti-K was found with other atypical antibodies, while eight samples contained only anti-K immune antibody. Four of the eight anti-K sera agglutinated saline-suspended cells; but the reactions in most cases were weak, and in none of the eight was the saline titre found to be higher than 1:1. The indirect Coombs technique (antiglobulin) gave titres approximately equal to those found by the use of AB serum, both as the diluent and as the cell-suspending medium. Incompatibility between eight anti-K sera and K-positive cells from six persons was detected equally well by the indirect Coombs test and the serum (direct centrifugation) technique. The incompatibilities were shown with fresh cells, and also with cells three weeks old; but the reactions were weaker with old cells. It was considered that bovine albumin tended to decrease or destroy the reactions in most cases.

Levine, Backer, Wigod and Ponder (1949) described a blood group property "Cellano", present in 99.8% of 2500 blood samples. The property was subsequently shown to be allelomorphous to K, and was called k. The antibody occurred in a mother with an infant which suffered a mild form of hæmolytic disease. Tests were performed at 37° C. with cells suspended in saline. The agglutinating antibody showed little loss of activity over a period of eighteen months.

Race and Sanger (1954) calculated the Kell genotype frequencies in England as follows:

$$KK = 0.0021$$

$$Kk = 0.0872$$

$$kk = 0.9107$$

Family studies indicate that the K antigen depends on a gene capable of expressing itself in single or in double dose.

From the foregoing summary of anti-K cases recorded in various countries, it becomes apparent that thought must be given to possible Kell immunization as well as to Rh immunization in pregnancy, and that when patients receive multiple transfusions of blood over an extended period they may produce anti-K. The present paper, which tabulates a series of cases in which anti-K was found, indicates that Australian laboratories have been on the lookout for such cases. In each instance identification of the antibody was made either in the original laboratory or in Melbourne.

#### Materials and Methods.

The methods commonly used in Australia for detecting anti-K and other atypical antibodies are as follows:

#### Agglutinating Antibodies.

Tests are performed on glass slides three inches square divided into 16 squares. To one drop of the patient's

undiluted serum is added one drop of a 10% suspension of the test cells in glucose-citrate solution (Simmons, Graydon, Semple and Taylor, 1951), and the two are mixed thoroughly by rotation. If a titre is to be determined, saline dilutions of the patient's serum (1/5, 1/10, 1/20 *et cetera*) are made and tested in the same way. Tests are performed at both 20° C. and 37° C., and the results are read after thirty to sixty minutes.

#### Blocking Antibodies.

To one drop of the patient's serum is added one drop of a 10% suspension of the test cells in 30% bovine albumin, and the two are mixed thoroughly. If a titre is to be determined, the patient's serum is serially diluted as described above with normal group AB serum. Tests are performed at 37° C., and the results are read after sixty minutes.

#### Comment.

The glass slides are placed in moist chambers at both 20° C. and 37° C.; these chambers consist of six-inch Petri dishes containing moist asbestos filter pads, with small rods on which to place the slides. The slide technique has been condemned mainly by non-users on the grounds that evaporation may cause unreliable results by rouleaux formation; but slides placed in moist chambers show no evaporation even when examined after having stood for forty-eight hours, and results obtained are as reliable as with any other technique. Slides are easily kept clean (grease-free) with a soap and water wash, while the cleansing of large numbers of small tubes is often unsatisfactory, and is time-consuming.

The results of the agglutinating and blocking tests are read macroscopically, and negative results are checked with a hand lens. At the time of reading, the slides should be tilted very gently two or three times, and not vigorously rotated, which seems to be a natural action.

#### Antiglobulin Test (Coombs).

One drop of a 10% to 30% suspension of the test cells in saline or glucose-citrate solution is added to three drops of the patient's serum, and the mixture is incubated for one hour at 37° C. in Wassermann tubes. The red cells are washed three times and centrifuged with the tubes full of saline, and after the third wash enough saline is left or added to give a cell concentration of at least 10%. One drop of this suspension is added to one drop of suitably diluted Coombs reagent (antiglobulin serum) on a glass slide, and the two are mixed by rotation. The slide is placed in a moist chamber and examined with a hand lens for agglutination after five minutes and again at ten minutes.

#### Results and Discussion.

Details of 11 examples of immunization to the blood factor Kell are given in Table I.

Immunization was detected in seven Rh-negative and four Rh-positive individuals, of whom 10 were females and one was a male. Seven were of group O, three were of group A (A<sub>1</sub> and A<sub>2</sub>), and one was of group B.

The patient in Case IX, classed as Rh negative, was of Rh type rh<sup>+</sup>rh<sup>-</sup>, while her Kell-negative husband was of type rh<sup>+</sup>rh<sup>-</sup>. In each instance the absence of Rh<sub>0</sub> (D<sup>+</sup>) variants was proved by antiglobulin tests after sensitization of the cells with two pure and potent anti-Rh<sub>0</sub> (D) sera. This Rh combination in marriage would be rarely encountered. There are three young children of the marriage, but they have not been Rh typed. The patient had received about seven pints of blood between 1953 and 1954, and her immunization was due entirely to blood transfusions; but the Kell status of the blood used is not known.

In six instances specific anti-K was produced, while in five instances one or two extra atypical antibodies were also formed. In three cases anti-K was detected only in the blocking or incomplete form, while in eight instances both agglutinating and blocking antibodies were present.



TABLE I.  
Eleven Examples of Anti-Kell.

Case.	Detected or Submitted by.	A B O and Rh Group.	Atypical Antibodies Present.	Nature of Anti-K.	Method by which Anti-K was Initially Detected.	Detected following Pregnancy or Blood Transfusion.
I .. ..	R. Jakobowicz.	O rh.	K, Rh <sub>0</sub> , rh'.	Blocking.	Antiglobulin test.	Pregnancy and blood transfusions.
II .. ..	P. I. A. Hendry.	O rh.	K, Rh <sub>0</sub> , rh'.	Blocking.	Use of 30% bovine albumin.	Blood transfusions.
III .. ..	P. I. A. Hendry.	B rh.	K, Rh <sub>0</sub> , rh'.	Blocking.	Use of 30% bovine albumin.	Pregnancy and blood transfusions.
IV <sup>a</sup> .. ..	G. H. Vos.	O rh.	K, Fy <sup>a</sup> .	Agglutinating blocking.	Antiglobulin test.	Blood transfusions.
V <sup>a</sup> .. ..	J. A. Albrey.	A <sub>1</sub> rh.	K.	Agglutinating blocking.	—	Pregnancy.
VI <sup>a</sup> .. ..	N. Silvester.	ORh <sub>1</sub> rh.	K.	Agglutinating blocking.	Antiglobulin test.	Pregnancy.
VII .. ..	V. I. Krieger.	A <sub>2</sub> Rh <sub>1</sub> Rh <sub>2</sub> .	K.	Agglutinating blocking.	Antiglobulin test.	Pregnancy.
VIII .. ..	R. Jakobowicz.	ORh <sup>+</sup> .	K.	Agglutinating blocking.	Antiglobulin test.	Blood transfusions.
IX .. ..	E. A. Richards.	Orh <sup>+</sup> rh.	K.	Agglutinating blocking.	Saline agglutination test.	Blood transfusions.
X .. ..	R. Jakobowicz.	A <sub>1</sub> Rh <sub>1</sub> Rh <sub>2</sub> .	K, rh <sup>+</sup> .	Agglutinating blocking.	Saline agglutination test.	Blood transfusions.
XI .. ..	R. Jakobowicz.	ORh <sub>1</sub> rh.	K.	Agglutinating blocking.	Antiglobulin test.	Pregnancy.

<sup>a</sup> In Case IV the antibody was identified as anti-K by Mr. G. H. Vos of Perth. By using a slide test he obtained 37 positive results in 471 random samples of blood of white Australians (7.86%).

<sup>a</sup> In Case V the antibody was identified as anti-K by Dr. C. A. Holman, of London. He observed that the antibody reacted in saline, in albumin, and with antiglobulin.

<sup>a</sup> Miss N. Silvester, of Brisbane, obtained eight positive results in 80 random blood samples (10%) by the antiglobulin test with this serum.

It was first thought that anti-K occurred only as a blocking serum. However, Dunsford (1949) reported finding the first example of agglutinating anti-K, and we were able to test a sample of this serum about that time. In the present series, detection of eight of the 11 anti-K sera was possible by the slide technique, when the undiluted serum was mixed with a 10% Kell-positive cell suspension, and the result was read after the mixture had stood for thirty to sixty minutes at room temperature (20° C.). An unexpected observation has been that while the slide test may clearly indicate a positive reaction, parallel tests by the conventional tube technique may fail to show any evidence of agglutination. We are grateful to Dr. R. R. Race for confirming this observation with an agglutinating anti-K serum sent to him from Melbourne.

Two of the eight anti-K sera have been used for routine K testing by the slide method, and have given comparable results in extensive parallel tests with other anti-K sera by the antiglobulin technique. It has been noticed with some anti-K sera that Kk cells give a weaker reaction than KK cells. While eight examples of anti-K with some agglutinating antibodies have been demonstrated, and while two of these sera give good slide agglutination in tests with undiluted serum, in no case has there been any saline agglutinating titre beyond a dilution of one in two. The blocking titre in five of the eight cases has been about one in 20 or higher. The most effective temperature for demonstrating anti-K agglutinating activity in all of the eight sera was about 20° C., rather than 37° C., as is generally thought.

It will be noticed that the methods by which anti-K was initially detected varied (antiglobulin, 30% bovine albumin or saline agglutination) according to the laboratory and the procedures in use, and to whether the tests were being performed as blood compatibility tests or, in some cases, as routine antiglobulin tests on the newborn. From our experience it would seem fair to say that the antiglobulin test would detect anti-K in all 11 instances, and that some saline (glucose-citrate) agglutinating activity was detectable at 20° C. on slides in eight out of 11 cases. In two instances the initial detection was made by the use of cells suspended in 30% bovine albumin, in compatibility tests performed at 37° C. Unfortunately, all the 11 anti-K sera were not further tested at the time of detection with K-positive cells suspended in 30% bovine albumin; but as two of the weakest K-reacting sera were found by this method, it seems possible that all 11 would react under our conditions of testing.

Case X, in which anti-K and anti-rh<sup>+</sup>(E) were produced as the result of blood transfusions in an Rh-positive woman, is of interest in that blood shown to be compatible by the antiglobulin test caused a mild transfusion reaction. The same blood in saline, or in 30% bovine albumin, tested against the pre-transfusion serum sample, was incompatible, owing to the presence of agglutinating anti-rh<sup>+</sup>(E) in the patient's serum. The findings were confirmed in two laboratories. It was assumed that linkage between rh<sup>+</sup>(E) and the corresponding antibody was such that normal saline washing employed in the antiglobulin test broke the antigen-antibody combination. Two examples have now been seen in Melbourne in which the antiglobulin test failed to demonstrate an incompatibility to the blood antigen rh<sup>+</sup>(E). These failures indicate that no one blood compatibility test alone is infallible, even when such a sensitive test as the antiglobulin technique is employed.

In those cases in which the Kell antibody occurs as the only atypical antibody, it may cause hemolytic disease of the newborn or, if undetected, blood transfusion reactions. In the present series, in which anti-K was present alone in six instances, it was responsible for four cases of hemolytic disease, with rapid death in one case due to hydrops. In two cases anti-K was detected prior to blood transfusion, once by saline agglutination, and once by the antiglobulin test.

#### The Kell (K) and Duffy (Fy<sup>a</sup>) Antigens in White Australians.

In 640 random blood samples tested with anti-K by the slide agglutination technique at 20° C., it was found that 60 (9.375%) were Kell-positive. About half the tests were controlled by the antiglobulin technique, another anti-K serum being used.

In 660 random blood samples tested with anti-Fy<sup>a</sup> by the antiglobulin technique, 429 (65%) were Duffy-positive. The anti-Fy<sup>a</sup> serum used had been generously supplied by Dr. A. S. Wiener, of New York. The foregoing results include those previously given by Simmons, Graydon and Semple (1953), who found the incidence of Fy(a+) to be 70%, and that of K+ to be 9% in 100 white Australians selected at random.

#### Antigenicity of the K Factor.

Grundorfer (1955) points out that Mollison and also Sanger *et alii* have suggested that the Kell factor is almost as antigenic as the Rh<sub>0</sub> factor, while Rosenfield and Vogel

compare its potency with the factor  $hr'(c)$ . Wiener, Brancato and Waltman (1953) discussed the antigenicity of the antigens  $Rh_0$  and K and concluded on clinical evidence that K was less antigenic than  $Rh_0$ . Nine examples of anti-K have been identified in Melbourne, and during the same period there have been 13 examples of anti- $hr'(c)$  and 16 of anti- $rh''(E)$ . It is generally accepted, and our own experience over years bears this out, that  $Rh_0$  (D) is the most antigenic, and next in activity is  $rh''(C)$ .

Graydon (1955) has generously permitted publication of the results of calculations made by him which are concerned with these five blood factors, and the relative frequencies expected if all factors are equally antigenic, for the first, second and third pregnancies, and also for the first, second and third random blood transfusions.

Table II shows the expected frequencies in which conditions are suitable for antibody development, and in addition the expected figures in each case relative to the value  $Rh_0$  (D) = 100. It will be seen that the expected frequencies differ for successive pregnancies, and for successive blood transfusions, and that the expected frequencies in second and third pregnancies differ from those in second and third blood transfusions. It should be noted that the examples found (13 anti- $hr'$ , 16 anti- $rh''$  and nine anti-K) are the result of immunizations by either pregnancies, or blood transfusions, or both, and the proportions are generally of the same order as the expected frequencies seen in the first pregnancy and first transfusion column (Table II). However, the proportions are very

TABLE IIA.

Antigenicity of Blood Factors; Relative Frequencies Expected Due to Pregnancy if Blood Factors are Equally Antigenic.

Factor.	First Pregnancy.	Second Pregnancy.	Third Pregnancy.
$Rh_0$ (D) .. ..	0.0919 100	0.0662 100	0.0477 100
$rh''$ (C) .. ..	0.1357 148	0.0877 132	0.0566 118
$rh''$ (E) .. ..	0.1143 124	0.0622 94	0.0388 71
Kell (K) .. ..	0.0462 50	0.0237 36	0.0121 25
$hr'$ (c) .. ..	0.1095 119	0.0750 113	0.0513 108

different from those seen in the second and third blood transfusion column. If comment can be made on this mixed series, it is that the factors  $rh''$ ,  $hr'$  and K, in sensitizing patients by pregnancy, by blood transfusion or by both, have produced numbers which, when viewed in the light of the expected frequencies given in the third pregnancy and third transfusion columns of Table II, suggest that the order of antigenicity is K,  $rh''$  and  $hr'$  respectively, K being the most effective of the three in stimulating antibodies.

From the frequency of occurrence of the relative phenotypes in the normal population, we should expect to find anti- $rh''(C)$  and anti- $rh'' + Rh_0$  (C + D) in approximately equal numbers; but over the above-mentioned period we have encountered a large number of anti- $rh'' + Rh_0$ , and only two possibly monovalent anti- $rh''$  sera. Thus, the presence of the  $Rh_0$  antigen influences the apparent effectiveness of the  $rh''(C)$  antigen, in that a mother's possession of  $Rh_0$  as in  $Rh_0$  not only prevents her from forming  $Rh_0$  antibodies in response to the simultaneous introduction of  $rh''$  and  $Rh_0$  antigens, but it makes less likely her development of  $rh''$  antibodies. Thus, there appears to be some relationship between these two antigens.

In pronounced contrast is the relationship of the  $Rh_0$  (D) and  $rh''(E)$  antigens. No such interference from  $Rh_0$  present in the  $Rh_0$  mother appears to operate in regard to the  $rh''$  antigen, for we would expect anti- $rh''(E)$  to occur nearly three times as frequently as anti- $rh'' + Rh_0$  (E + D), and the observed ratio is certainly no less than this figure. The low frequency of anti- $rh'' + Rh_0$  suggests that the introduction of  $Rh_0$  antigen along with  $rh''$  antigen tends to prevent the formation of anti- $rh''$  in Rh-negative individuals.

These contrasting effects may be summarized as follows: (i) The  $Rh_0$  antigen native to the recipient tends to prevent formation of anti- $rh''$  in that individual, but does not exert this suppressive effect on the formation of anti- $rh''$ . (ii) The  $Rh_0$  antigen introduced into Rh-negative individuals does not prevent the formation of  $rh''$  antibodies, but tends to suppress the formation of  $rh''$  antibodies. These two activities may be manifestations of the preeminent position of the  $Rh_0$  (D) antigen in Rh serology.

TABLE IIB.

Antigenicity of Blood Factors; Relative Frequencies Expected if Factors are Equally Antigenic and Sensitization is Due Only to Blood Transfusion (Random).

Factor.	First Transfusion.	Second Transfusion.	Third Transfusion.
$Rh_0$ (D) .. ..	0.0919 100	0.0563 100	0.0345 100
$rh''$ (C) .. ..	0.1357 148	0.0614 109	0.0278 81
$rh''$ (E) .. ..	0.1143 124	0.0187 33	0.0030 8.7
Kell (K) .. ..	0.0462 50	0.0024 4.3	0.00012 0.3
$hr'$ (c) .. ..	0.1095 119	0.0600 106	0.0328 95

#### Labiality of the K Antigen.

There is some confusion as to the stability of the Kell antigen in comparison with other blood group antigens. Sanger, Walsh and Kay (1951) reported that two clotted blood samples, sent on a three-day air trip without refrigeration, failed to give positive reactions with an anti-K serum by the antiglobulin technique. Race and Sanger (1954) state that on occasion they have failed to detect the K antigen by the use of a saline test, and even when using an antiglobulin test on twenty-four-hour-old finger-prick samples. Simmons, Graydon, Semple and Kodama (1953) reported testing 12 blood samples from persons of known types on their panel, and these samples had been stored in glucose-citrate preserving solution at 5° C. for periods from five days to twenty months. The Rh, Kell and Duffy tests were entirely satisfactory, and the results obtained were identical with those found with freshly collected cells. From these and other tests the conclusion was reached that most of the blood group antigens of suitably preserved cells deteriorate at much the same rate. In tests with potent sera this deterioration was regarded as unimportant; but when sera are weak it may seriously affect the accuracy of the results. Earlier it was pointed out that Grove-Rasmussen *et alii* (1954) showed incompatibility between K-positive cells from six persons and eight anti-K sera both with fresh blood and with blood which had been stored for three weeks. In blood transfusion practice, detection of the K antigen in compatibility testing should not present any real difficulties when the patient possesses anti-K.

#### Summary.

- Details are presented of 11 instances of immunization to the blood factor Kell. Nine of these examples were initially identified in Melbourne. Anti-K may cause haemolytic disease of the newborn or blood transfusion reactions.
- The K antigen has proved to be an effective antigen, and thought must be given to it as well as to Rh in immunization problems.
- The labiality and antigenicity of the K antigen have been briefly discussed.
- It was found that 60 of 640 (9.375%) random blood samples from white Australians were Kell positive, while 429 of 660 (65%) were Duffy positive.

#### Acknowledgements.

The author expresses gratitude to all who generously cooperated in this work, and offers special thanks to Dr. Rachel Jakobowicz, of the Red Cross Blood Transfusion Service, and to his colleague, Dr. J. J. Graydon.

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ÆSCULAPIUS, THE STAFF AND THE SERPENT IN ROME.

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IF on a visit to Rome you go down the *Via Arenula*, you will come to the river Tiber (*Fiume Tevere*). Do not pass over the *Ponte Garibaldi*, but turn aside and you will see the island in the Tiber (*Isola Tiberina*).

There, over two thousand years ago, in 293 B.C., was founded the forerunner of the first public hospital. If you are interested sufficiently to cross to the isle and over the *Ponte Fabricio* (62 B.C.), you will see the Hospital of Saint John of God (*San Juan de Dios*) and also the Church of Saint Bartholomew of the Island. They stand where stood in ancient times the Temple of Æsculapius, the Greek god of medicine.

Then seek out the aged caretaker of the church. His eyes will lighten at the mention of Æsculapius of Epidaurus and later Rome.

In the environs of the Church of Saint Bartholomew, with penthouses on one side and the Tiber on the other, lies a shady trellised pathway. Down the trellised path the caretaker will lead you, down the time-worn steps to the river bank, and then he will proudly point to the ruins—building stones hewn of travertine.

Drawing near, you will see the staff and the serpent of Æsculapius—the symbol of the medical profession—sculptured on the travertine. It would appear that with the passage of time, although the hewn stones have become worn by the elements, the staff and the serpent stand more eminently in relief. The head and shoulders of Æsculapius are also to be seen sculptured alongside the symbol of medicine.

Æsculapius was the legendary Greek god of medicine, being the son of the Greek god Apollo (inventor of the healing art) and the nymph Coronis. He was probably

born in Epidaurus in Greece, some thirty miles from Athens. Hygeia (the goddess of health) and Panacea (the healer of all ills) were two of his daughters, and Telesphorus, the boy genius of healing, was probably his son. He was educated by the centaur Cheiron, who was generally regarded as the founder and master of medicine, especially surgery.



FIGURE I.

The island in the Tiber and the river bank. The arrow indicates all that remains of the Æsculapian temple.

The cult of Æsculapius almost certainly originated in Thessaly and was introduced into Athens in 429 B.C. Epidaurus soon became the centre of the cult, and temples were erected to Æsculapius in many parts of Greece near healing springs or on high mountains. Those coming to the temples underwent a preliminary period of treatment consisting of purgation, baths, and abstinence from rich



FIGURE II.

A close view of the building stones, showing the staff and the serpent, and also the head and shoulders of Æsculapius on the adjacent stone.

foods and wines. After this came several days of strict diet, and the ailing were admitted to the temple. Then only were they admitted to the ceremony proper of the Æsculapian cult, with suggestive prayers by the priests and one or more nights at the statue of Æsculapius awaiting the healing dream. Sometimes the priest, in the mask of the Epidaurian god, performed treatments in the form of suggestion or of interpretation of the dream, or prescribed remedies. The suggestion therapy during the hypnotic state, together with the healthy, carefully chosen sites of the temples and the general care of the ailing, resulted in many receiving much benefit from their visit to the temple. All those who were healed offered a sacrifice, especially a cock, and also hung up votive tablets on which were recorded their names and the nature of the cure.



In the temples were kept tame serpents. They had long been regarded as a symbol of prudence and rejuvenation by the shedding of their skins, and were believed to have the power of discovering curative herbs. They were also believed to be guardians of the wells of salutary powers. (The sacred serpent of Æsculapius was the *Coluber longissimus*, bronze in colour and up to six feet in length. It is practically extinct today.)

The advent of the Æsculapian cult to Rome took place some three hundred years before Christ (293 B.C.). Being unable to check "a dire pestilence" (plague), the rulers of ancient Rome sought the counsel of the Sibylline Books, and were directed to bring Æsculapius from Epidaurus to Rome. A galley was sent to the Saronic Gulf, and an envoy visited the temple at Hieron (near Epidaurus).



FIGURE III.

A closer view of the staff and the serpent, which stands about three and a half feet in length.

According to Ovid, the Epidaurians were not at all willing to help the Romans, wishing to retain the god for themselves. However, the galley returned with Æsculapius in the form of one of the sacred serpents. The galley entered the Tiber with the serpent on the mast top, and touched land where the river embraces the *Isola Tiberina*. The sacred serpent at once left the ship and found refuge on the island. From that moment the plague rapidly abated.

In gratitude to Æsculapius, now among them in the form of a serpent, the island was modelled into the shape of a great galley, and a temple was erected where this day stands the Church of Saint Bartholemew, and on the site of the ancient abaton now stands a hospital, the Hospital of Saint John (*San Juan de Dios*). A well existing on the island became sacred to Æsculapius, and from that day of the advent of Æsculapius to Rome for over 2200 years, the *Isola Tiberina*, through pagan and Christian times alike, has been devoted to the care and succour of the ailing.

A small portion of the stern of the hewn stone galley still exists with the remains of the image of Æsculapius and the effigy of the staff and serpent. (In his youth Æsculapius was shut up in the house of Glaucus, whom he was to cure. Whilst he was standing in thought, a serpent came and twined itself about his staff. He killed the serpent, but yet another came carrying a herb and brought it back to life. It is said that Æsculapius used this herb with similar effect on man.)

There being no provision for the slaves of the Romans when they were ill, it became customary to send the ailing and the worn-out slaves to the island; so the island first became a refuge for the sick poor of Rome, and indeed, it has maintained such a service right up to this present time.

Thus we have the Temple of Æsculapius founded in 293 B.C. on the *Isola Tiberina* in Rome as the forerunner of the public hospital system, and with its development it is appropriate that we find the adoption of the staff and the serpent of that Epidaurian god as the medical emblem.

## Reviews.

**Whys and Wherefores in Tuberculosis.** By George Day; 1955. London: The National Association for the Prevention of Tuberculosis. 8" x 5½", pp. 44. Price: 3s. 6d.

**A New Classification of Tuberculosis with New Diagnostic Standards.** By Milosh Sekulich, M.D., edited by H. Stanley Banks, M.D., F.R.C.P.; 1955. London: William Heinemann (Medical Books) Limited. 8½" x 5½", pp. 64, with many illustrations. Price: 3s. 6d.

Two very different booklets about tuberculosis have been issued recently. The first, "Whys and Wherefores in Tuberculosis", by Dr. George Day, is published by the National Association for the Prevention of Tuberculosis, and is designed to tell patients, as the title implies, some details of the many things that they would like to know concerning all aspects of the disease. It opens with an historical review of tuberculosis, and goes on to discuss the different forms of therapy, and so gives patients some idea of what they may be expected to undergo in order to regain their health. Physicians can refer their patients to this little book, for it is written in a very pleasing manner, and they will derive much benefit from it.

The second booklet is "A New Classification of Tuberculosis", by Dr. Milosh Sekulich, who is well known for his writings on the subject. It presents a system which would be of value to the statistician, but which is perhaps rather too complex for everyday use in the hospital clinic or the consulting room. To appreciate this new classification fully, it is necessary to read it in conjunction with the author's earlier work on the subject. Whereas this booklet is only of 63 pages, the other runs into some 356 pages, and is referred to frequently in the text of the booklet. It is problematical whether this very comprehensive classification will supersede those in present use.

In addition to the subject, both booklets have one thing in common—each is priced at 3s. 6d.

**A Short Textbook of Surgery.** By C. F. W. Illingworth, C.B.E., M.D., Ch.M., F.R.C.S. (Ed.), F.R.F.P.S. (Glas.), Hon. F.A.C.S.; Sixth Edition; 1955. London: J. and A. Churchill, Limited. 9½" x 6", pp. 636, with 227 illustrations. Price: 37s. 6d.

In "A Short Textbook of Surgery", now in its sixth edition, Professor Illingworth has been remarkably successful in giving the student a readable account of surgery, including its associated specialties, in a reasonably small volume. Each section provides a very good framework upon which to build. This book gives the undergraduate a much better chance of mastering the basic minimum than do those textbooks which are two or three times as large.

The author has "attempted to rationalise the clinical approach"; in this he has been very largely successful. It is, however, a pity that in that most common surgical disease, appendicitis, he explains the centre pain by reference to the body-wall, whereas in fact the pain is a deep pain, purely visceral and independent of the body wall and of its innervation.

In the fifth edition, the old barbarities of bowel management in appendicitis and peritonitis were given up. Let us hope that in the seventh edition they will also be given up before and after the operation for intestinal obstruction. That the mixed parotid tumour "shells out readily" is perhaps optimistic, in view of the fact that its tendency to recurrence has inclined surgeons to parotid lobectomy. Figure 114 is a diagram illustrating the operation for cure of *hallux valgus*. It shows the associated *metatarsus primus varus* cured by the operation, but it gives no indication as to

how this was done. The old Kanavel operation for paronychia is described, but no mention is made of the bloodless method of lifting the nail fold and, if necessary, snipping the nail margin.

**Glaucoma: A Symposium Organized by the Council for International Organizations of Medical Sciences, Established under the Joint Auspices of UNESCO and WHO,** edited by Sir Stewart Duke-Elder; 1955. Oxford: Blackwell Scientific Publications. 9" x 5½", pp. 362, with many illustrations. Price: 37s. 6d.

This publication edited by Sir Stewart Duke-Elder is a record of papers and discussions which were presented at a symposium on glaucoma organized by the Council for International Organization of Medical Services. After an introduction by the editor, the book is divided into seven parts.

Part one on anatomy, physiology and pathology is contributed by Duke-Elder and Ashton, part two on the dynamics of the intraocular fluid is by Langham and Kinsey, part three on resistance to outflow of the aqueous is by Bárány, part four on gonioscopy is by François, part five on provocative tests is by Leydhecher, Kronfeld and Duke-Elder, part six on clinical aspects is by Weekers and Hodgson, and part seven is a general discussion with conclusions by Friedenwald and Duke-Elder.

All will agree that a first-class team had been assembled and a wide range of subjects listed for discussion. The reader will not be disappointed with the presentation. Although there is no pretence that this is a comprehensive book on the subject, perusal of the contents leaves one with the feeling that at least there could be very little left to know about primary glaucoma.

An interesting conclusion of the members of the symposium is that they agreed that primary glaucoma should be classified as "closed angle glaucoma" and "simple glaucoma" and that the term open angle glaucoma ought to be discarded.

The book contains a wealth of information and should be in the library of all practising ophthalmologists.

**The Mentally Retarded Child: A Guide for Parents.** By Abraham Levinson, M.D.; edited with a preface to the British edition by Kay McDougall; introduction by Pearl S. Buck; 1955. London: George Allen and Unwin Limited. 8½" x 5½", pp. 128. Price: 12s. 6d.

This book is written as a guide for parents who have the misfortune to have a mentally retarded child. Whilst it was primarily written to suit circumstances in the United States, this British edition has been edited by Kay McDougall, and so is somewhat suited to conditions in this country.

As the author is Professor of Pediatrics, Northwestern University Medical School, and also Director of the Dr. Julian D. Levinson Research Foundation, named in honour of his son and devoted to the study of the problem of mentally retarded children, it can be seen that he has a wide practical knowledge of all aspects of the subject. The book puts forward a series of ten commandments for parents, which include the following:

Don't adopt a defeatist attitude, don't develop a complex of shame or guilt, don't pauperise yourself to give your child the best, and don't be afraid to have other children.

The tragic impact of a retarded child on a family is well known to all doctors, and they can recommend this book to parents of such children, for it will give them a great deal of practical advice to help them overcome a problem, which, if not tackled sensibly, can disrupt their whole social and family life.

**Forensic Medicine: A Textbook for Students and Practitioners.** By Sir Sydney Smith, C.B.E., LL.D., M.D. (Edin.), F.R.C.P. (Edin.), D.P.H., F.R.S.E., Hon. M.D. Louvain, and Frederick Smith Fiddes, O.B.E., M.D.; Tenth Edition; 1955. London: J. and A. Churchill Limited. 9½" x 6", pp. 656, with 173 illustrations. Price: 40s.

This book in its tenth edition deals with the usual forensic subjects, violent and unexpected death, wounds, rape, abortion, infanticide, identification of human remains, accidents and compensation, poisons *et cetera*, and the various laws that concern the medical practitioner and his legal responsibilities.

While the general principles remain much the same, this edition reflects modern advances and research which have obviously shaken some of the more traditional teaching. For

example, we find an alternative set of tables included for calculating the height of the skeleton from dead long bones. Pearson's tables so long relied upon have often proved fallible.

Theoretical discussions are not prominent in the text, and in general the whole subject is dealt with in a straightforward and practical manner. Although there is joint authorship, an individual opinion is frequently given.

As the general practitioner is expected in this country to carry out post-mortem examinations for the coroner, and as the great majority of these deaths are natural in origin, he needs in a reference text-book a full discussion of unexpected death from natural causes, more than is given in this work. This deficiency could perhaps be made good at the expense of the chapter on the differential diagnosis of coma.

Rupture of the heart wall, especially with regard to infarction, we find is common, nor do we find fracture of the sternum so uncommon. Experimental work shows that Schäfer's method of artificial respiration is not so satisfactory as has been thought and taught; other methods are to be preferred. When there is no air in the lungs, does the presence of Tardieu's spots on the lungs indicate attempts at respiration? In view of the recent doubts cast on the so-called asphyxial picture at post-mortem examination, the statement on page 249 is a good one. In the discussion on the biochemical changes in the blood in drowning, an indication of what normally happens to the chlorides *et cetera* after death would be valuable. There is a very good chapter on infanticide. The significance of hyaline membrane as an indication of legal live birth is doubted in this book.

After the ingestion of a fatal quantity of cyanide, there can be no doubt, as the authors point out, about the possibility of various actions during a period of consciousness, and death may not take place for some hours. The action of BAL in the body is well presented, and there is useful information about snake-bite.

The advice given to the medical practitioner as a witness and the presentation of his evidence in court is excellent. It is surprising just how much practical and useful information is packed into the pages of this book, which continues to serve a very useful purpose for reference and teaching.

**The Year Book of General Surgery (1955-1956 Year Book Series).** Edited by Evarts A. Graham, A.B., M.D.; 1955. Chicago: The Year Book Publishers, Incorporated. 7½" x 5", pp. 656, with 182 illustrations. Price: \$6.00.

With its broad survey of current surgical literature and its large section on anaesthesia, this Year Book should have a wide appeal. As the editor, Evarts A. Graham, points out in an introduction, surgery is undergoing very profound changes. Many formerly frequent conditions have almost disappeared or have passed out of the operative surgeon's hands; other conditions have become amenable to surgery because of new surgical and ancillary techniques; more venturesome surgery has become possible in the management, for example, of cancer, although the wisdom of its use is seriously questioned. All these changes are reflected in the current volume of this Year Book. It is noted, for example, that of the 536 pages devoted to surgery, nearly one-quarter are taken up by surgery of the heart, lungs and mediastinal structures, and substantial space is allotted to "shock, fluids and electrolytes", nutrition, antibiotics and similar "general considerations". Most of the rest of the material is grouped on an anatomical basis with separate chapters on technical contributions, wounds and wound healing, neoplasms, hypertension, pillo-nidal cysts and hernia.

The section on anaesthesia, edited by Stuart C. Cullen, has chapters on depressant drugs, ventilation, inhalation anaesthesia, muscle relaxants, barbiturates, spinal anaesthesia, regional anaesthesia, hypothermia and hypotension.

**Spot Diagnosis: With Notes on Therapy.** Compiled by the editors of "Medicine Illustrated"; 1955. London: Harvey and Blythe, Limited. Volume II. 8½" x 5½", pp. 128, with 100 illustrations. Price: 8s. 6d.

This is the second volume to appear under this title, and like its predecessor consists of over 100 illustrations of various clinical conditions which are characteristic enough to suggest the diagnosis. The answer to the conundrum with short notes on the conditions illustrated appears on the reverse side of the page. The value of such a book in augmenting the experience of students, even though at second hand, is quite obvious, and the pictorial method needs no vindication these days. It will provide a corrective



for those students, still regrettably far too numerous, who labour under the delusion that there is more in books than in the wards. Its simple style and small size make it an ideal companion at the bus stop and allow a good deal of revision to be done quickly. The latter and smaller section of the book gives short but valuable summaries of treatment of some of the conditions illustrated. In brief, this is a pictorial "digest" of medicine which may help the student to spend a few spare minutes profitably.

**My Duodenal Ulcer and I.** By Stuart Morton; 1955. London: Christopher Johnson. 8½" x 5½", pp. 214. Price: 16s.

THE author of this book tells his tale in a straightforward and simple way. He first manifested symptoms of duodenal ulcer when he was a lad of eighteen years. He graduated in medicine at Glasgow in April, 1920. From this time on his tale is one of recurring melæna with operative treatment; gastro-jejunostomy was one of the earlier adventures. He spent most of his life as a ship's surgeon and his ulcer perforated when he was crossing the Atlantic Ocean. Eventually he submitted to partial gastrectomy which was not successful. Our author insists that operation is only an incident in the medical treatment of the condition. His observations on duodenal ulcer and its treatment are, as one would expect, sound, and repay perusal. Incidentally, his views on ships' surgeons and their work are worth while. He attained comparative peace after he married a woman who was a tower of strength to him. This book is worth reading because the author shows throughout the kind of temperament that goes with duodenal ulcer.

## Notes on Books, Current Journals and New Appliances.

**The Pocket Prescriber and Guide to Prescription Writing.** By Alistair G. Cruikshank, F.R.C.P.E.; Sixteenth Edition; 1955. Edinburgh and London: E. and S. Livingstone, Limited. 4" x 2½", pp. 299. Price: 5s.

THIS pocket-sized book contains hints on prescribing, a selection of prescriptions and notes on treatment of various diseases, a dosage table, and various pieces of information relating more particularly to treatment. If used as an *aide-memoire* rather than a text-book of treatment, it should be acceptable. It is a little surprising to find the antibiotics included in a chapter on the administration of sulphamides. Presumably this is an error in the layout of the book.

**Basic Surgical Skills: A Manual with Appropriate Exercises.** By Robert Tauber, M.D., F.A.C.S.; 1955. Philadelphia and London: W. B. Saunders Company, Melbourne: W. Ramsay (Surgical), Limited. 11" x 8½", pp. 82, with 51 illustrations. Price: £1 17s. 16d.

THIS book is based on the sound principle that the technique of the surgical elements which compose each single step of an operation must be completely mastered before any work in the operating room can be accomplished. To help the surgeon to train his fingers, the author has designed a "training board". This is essentially a wooden board, eleven and a half by twelve inches in size, furnished with a number of holes in which to fasten hooks, nails, rods and rings. Gauze pads also are fastened to it to be used as "tissue". With this simple but ingenious piece of apparatus the many exercises in the book can be carried out with advantage. The book is written in a straightforward fashion and is well illustrated. It will be most useful to any medical practitioner who wishes to acquire surgical skill and is prepared to devote to it the necessary time and patience.

**Mental Health and Infant Development: Proceedings of the International Seminar held by the World Federation for Mental Health at Chichester, England.** Edited by Kenneth Soddy, M.D.; 1955. London: Routledge Kegan Paul, Limited. Volume I, papers and discussions, 8½" x 5½", pp. 327, price 25s. Volume II, case histories, 8½" x 5½", pp. 294, price 25s.

THE first volume of this book contains the proceedings of a seminar on mental health and infant development held at Chichester in England in July and August, 1952. The second volume is made up of case histories, and contains approximately one-half of the case material specifically prepared for use at the seminar. The seminar was organized by the World Federation for Mental Health with support from the

World Health Organization and a number of other bodies. The members of the seminar came from all over the world, and included many people of eminence in the field concerned. The major subjects under which the seminar was conducted were child development patterns, studies of infant relationship formation, different cultural patterns and technological change, social and community provisions for mental hygiene, techniques for changing social practices, studies in psychology and neurology, and aids to education. Participants in the discussions came from many professional disciplines, and they brought many points of view to the discussion of an important subject.

## Books Received.

[The mention of a book in this column does not imply that no review will appear in a subsequent issue.]

**"The Surgical Clinics of North America."** Philadelphia Number: 1955. Philadelphia and London: W. B. Saunders Company. Melbourne: W. Ramsay (Surgical), Limited. 9" x 6", pp. 298, with many illustrations. Price: £8 2s. 6d. per annum with cloth binding and £6 15s. per annum with paper binding.

Consists of a symposium on applied physiology in modern surgery. There are 19 chapters and 33 contributors.

**"Microbiology: With Applications to Nursing"**, by Catherine Jones Witton, M.A. Second Edition; 1956. New York, Toronto, London: The Blakiston Division, McGraw-Hill Book Company, Incorporated. 9" x 6", with many illustrations; pp. 634. Price: \$6.00.

Intended for students in schools of nursing and other health fields.

**"A Guide to Psychiatric Books: With Some Suggested Reading Lists"**, by Karl A. Menninger, M.D. The Menninger Clinic Monograph Series Number 7; 1956. Second Revised Edition; 1956. London and New York: Grune and Stratton. 5½" x 8½", pp. 173. Price: \$4.75.

Written in answer to a demand for a checklist and guide to the literature of psychiatry.

**"A Practical Handbook of Psychiatry for Students and Nurses"**, by Louis Minski, M.D., F.R.C.P., D.P.M. Third Edition; 1956. London: William Heinemann (Medical Books), Limited. 7½" x 5", pp. 152. Price: 7s. 6d.

Methods of treatment are described from the results of personal experience.

**"A Handbook of Medical Hypnosis: An Introduction for Practitioners and Students"**, by Gordon Ambrose, L.M.S.S.A., and George Newbold, M.B., B.S., M.R.C.S., M.M.S.A., D.R.C.O.G., D.C.H., with forewords by William Moodie, M.D., F.R.C.P., D.P.M., and William S. Kroger, M.D.; 1956. London: Baillière, Tindall and Cox. 8½" x 5½", pp. 268. Price: 21s.

"A Handbook and not a text-book."

**"The Role of Algae and Plankton in Medicine"**, by Morton Schwimmer, M.D., and David Schwimmer, M.D., F.A.C.P.; 1955. New York and London: Grune and Stratton. 8½" x 5", pp. 95. Price: \$3.75.

Stress is laid on the great medical potential of algae and plankton.

**"Shock and Circulatory Homeostasis: Transactions of the Fourth Conference, December 6, 7 and 8, 1954, Princeton, N.J."** Edited by Harold D. Green, M.D.; 1955. New York, N.Y.: The Josiah Macy, Jr. Foundation. 9" x 6", pp. 291, with 69 illustrations. Price: \$5.00.

Divided into seven sections, in which a different subject is discussed, first by a principal speaker and then by others.

**"Midwifery: Principles and Practice for Pupils Midwives, Teacher Midwives and Obstetric Dressers"**, by R. Christie Brown, M.B., M.S., F.R.C.S., F.R.C.O.G., Barton Gilbert, B.Sc., M.D., F.R.C.S., F.R.C.O.G., Donald B. Fraser, M.A., B.M., B.Ch., F.R.C.S., F.R.C.O.G., and Richard H. Dobbs, M.D., F.R.C.P.; Fourth Edition; 1956. London: Edward Arnold (Publishers), Limited. 7½" x 4½", pp. 896, with 223 illustrations. Price: 25s.

The book "lays great stress upon physiological principles and their application to midwifery".



## The Medical Journal of Australia

SATURDAY, MAY 26, 1956.

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### SOLITUDE, LONELINESS AND SUICIDE.

Two thousand years ago Cicero put into the mouth of Scipio Africanus the famous phrase: "I am never less alone than when alone." It has since been echoed and counted as the mark of wisdom by many others; but there are dissenting voices. That gregarious soul Samuel Johnson held the view that "the solitary mortal is certainly luxurious, probably superstitious, and possibly mad". Francis Bacon opened his essay "On Friendship" with a critical comment on a sentiment of Aristotle:

It had been hard for him that spake to have put more truth and untruth together in few words than in that speech, "Whosoever is delighted in solitude, is either a wild beast or a god": for it is most true, that a natural and secret hatred and aversion towards society in any man hath somewhat of the savage beast; but it is most untrue that it should have any character at all of the divine nature, except it proceed, not out of a pleasure in solitude, but out of a love and desire to sequester a man's self for a higher conversation. . . .

By making this exception, of course, Bacon solves the seeming contradiction. Man is essentially gregarious and naturally enough regards the deliberate recluse with curiosity or suspicion. It is not for nothing that solitary confinement is a dreaded form of punishment, or that "It is not good for the man to be alone" has echoed down from Eden. The misanthrope is to be pitied if not feared. On the other hand many feel the need of solitude from time to time in order to take stock of themselves, to find repose of mind, to pause in the hectic business of living. This is good and necessary. Those who shun it vigorously, unless they are merely shallow, are in their turn to be pitied. They dread being alone with themselves; and it is not

surprising that they often prefer, as an alternative, to get drunk.

Solitude then does not necessarily mean loneliness. For those who are running away from themselves it brings a surfeit of undesired company. "Indeed, though in a Wilderness", writes Sir Thomas Browne, "a man is never alone, not only because he is with himself and his own thoughts, but because he is with the Devil, who ever consorts with our solitude, and is that unruly rebel that musters up those disordered motions which accompany our sequestered imaginations." But for those who use it wisely solitude brings peace and fresh vision for living. For those with a well-stocked mind it brings the good company of worthy things long cherished. For many it is, as W. S. Landor has said, "the audience-chamber of God". Aloneness of a man's body need not touch his spirit.

Loneliness, on the other hand, is essentially a thing of the spirit. It can be at its worst in the middle of a crowd. The best and wisest of men have known its desolation, although it may be possible to rise above it while still enduring it. This is the thought that Bernard Shaw brings out in his play *Saint Joan*, when Joan, realizing that the dignitaries of France have cast her aside, expresses herself frankly:

Do not think that you can frighten me by telling me that I am alone. France is alone; and God is alone; and what is my loneliness before the loneliness of my country and my God? I see now that the loneliness of God is His strength. . . . Well, my loneliness shall be my strength too. . . .

It is indeed the lot of all leaders to be alone; they can never be great or even adequate in their leadership unless they accept this and remain strong in their loneliness.

The tragic situation is that of those who meet loneliness without the resources to cope with it. It may be genuine loneliness; or it may be an abnormal state of mind, in which the individual wrongly feels himself to be cut off from his surrounding world. The outcome is much the same; and one of its fruits is attempted suicide. T. M. Ling, a psychiatrist, has been reported<sup>1</sup> as saying, at a Town and Country Planning Summer School held at the University of St. Andrews last year, that loneliness was one of the major social evils of our urban civilization and directly linked with the highest incidence of suicide. In London, suicides occurred to a greater extent in Holburn, Kensington, Chelsea and other areas where most people lived alone. Figures were significantly lower in poorer areas. The suicide-prone areas of Kensington, Bloomsbury and Marylebone harboured many transients, while often the type of housing had been changed by economic circumstances from high Regency and Victorian single family units to dwellings for a series of isolates, who led a lonely and dreamy existence in a single room. Ling suggested that in new housing estates town planners could avoid creating conditions that would contribute to such individual and family loneliness. At the same time it is to be remembered that those who are not physically separated from their relations and friends may yet, for a variety of reasons real or fancied, feel a terrible isolation. Their minds look out through the iron bars of a self-constructed prison and see between themselves and their fellows a great gulf of misunderstanding, lack of apprecia-

<sup>1</sup> *The Times*, September 2, 1954.

tion, and frustration. Or, like a street vagrant on a winter's night, they flatten their noses against the outside of the window pane and watch with longing and despair the happy unheeding play of those who enjoy the light and warmth inside. Many then lack the stoicism to face the thought expressed by Dryden:

But we like sentries are obliged to stand  
In starless nights, and wait th' appointed hour.

It may seem to them infinitely sweeter to pluck what James Thomson calls "the rich dark clusters of the Vine of Death" as they hang over the wall of the barren Garden of Life. They turn to death while they cry out for life.

According to E. Stengel and N. G. Cook,<sup>1</sup> there is reason for assuming that most suicidal attempts have a hidden "appeal character"—that is to say, "in the constellation of the patient's behaviour a call for help can be discerned". Commenting on Stengel's view of suicidal acts, "directed Janus-faced both towards a renewal of human relationships and towards death", I. R. C. Batchelor and M. B. Napier<sup>2</sup> describe it as a valuable formulation. Their own quite considerable data (compiled in Edinburgh), even where they do not confirm Stengel's findings and concepts, tend rather to qualify than to contradict them. However, they cannot confirm Stengel's statement that the suicidal attempt usually results in some modification of the individual's social situation with the initiation of a new era in his relationship to his environment. They found this to be so in certain instances, but only in a minority; sometimes the situation was made worse. Their view is that the appeal element is present most frequently and prominently in the suicidal attempts of certain temperamentally unstable individuals chronically in conflict with their society, but that it is doubtful if it is an element in all suicidal attempts. Moreover, an over-emphasis upon it may lead to an under-emphasis of the danger of suicidal attempts. This is reasonable enough. Many motives lie behind suicidal attempts; their elucidation in each individual case is of more value than generalizations. At the same time, the possible appeal character of a suicidal attempt and the loneliness in the mind of the person concerned need to be borne in mind by the doctor, usually the general practitioner and sometimes the young house surgeon, who makes the first contact with a would-be suicide after the event. Douglas Powers<sup>3</sup> has drawn vivid contrasting pictures in two case reports: in one the doctor was disdainful while efficiently repairing a self-inflicted neck wound, and the patient soon after made another attempt at suicide; in the other the doctor adopted an understanding attitude during a similar procedure, and the patient quickly gave him his confidence, with satisfactory results. Powers considers that it is a matter not so much of what the doctor says as of the attitude with which he regards and reacts to what has happened. A warm and accepting attitude prepares the way for the patient to talk. A wrong attitude may close the first and perhaps only avenue of assistance. This is true whether the patient is later referred to a psychiatrist or not. In this respect we may return to Francis Bacon for sound medical practice, whatever we may think of his *materia medica*:

A principal fruit of friendship is the ease and discharge of the fulness and swellings of the heart, which passions of all kinds do cause and induce. We know diseases of stoppings and suffocations are the most dangerous in the body; and it is not much otherwise in the mind; you may take sarza to open the liver, steel to open the spleen, flower of sulphur for the lungs, castoreum for the brain; but no receipt openeth the heart but a true friend, to whom you may impart griefs, joys, fears, hopes, suspicions, counsels, and whatsoever lieth upon the heart to oppress it, in a kind of civil shrift or confession.

This is a deep need of man. Princes in their exalted isolation will often, in Bacon's words, "raise some persons to be as it were companions, and almost equals to themselves, which many times sorteth to inconvenience". These are often called "favourites", but "the Roman name attaineth the true use and cause thereof, naming them 'participes curarum'". They become the sharers of cares. This is the role *par excellence* of the good doctor.

## Current Comment.

### "MYLERAN" AND CHRONIC MYELOCYTIC LEUCÆMIA.

Of the various malignant conditions to which the human body is prone, none has proved more resistant to treatment than the blood dyscrasias grouped under the name of leucæmia. Numerous therapeutic agents have been applied, including folic acid antagonists, radioactive circulating compounds, irradiation of bone centres, nitrogen mustards and steroid compounds. The effects have been generally disappointing, and in a recent survey A. Haut *et alii*<sup>1</sup> reported that there was no statistical evidence that any compound so far produced had any material effect on the survival time of patients with acute leucæmia. This finding is particularly discouraging when it is considered that, above all malignant conditions, the acute leucæmias are diseases of the young and of the active. The treatment of the chronic leucæmias has also been fraught with disappointment; but evidence is accumulating which indicates that in the case of chronic myelocytic leucæmia considerable progress has been made towards finding a safe substance which will control the disease.

A. Haut, S. J. Altman, D. E. Cartwright and M. M. Wintrobe<sup>2</sup> have studied the effects of "Myleran" in the treatment of chronic myelocytic leucæmia. "Myleran" is a sulphonic acid ester, and it has been used by the authors in the treatment of 16 patients. Each of these has received from one to six courses of this drug in a daily dose by mouth of from four to six milligrammes taken before breakfast. The duration of therapy was for the most part between forty-two and seventy-one days. Patients were ambulant during therapy, and were examined at intervals of from one to three weeks. Treatment was continued until desired clinical criteria were found, and treatment was restarted when relapse occurred. Often within the first two weeks the patients noticed an increase in appetite and a sense of well-being, and thereafter all but three patients remained free of symptoms despite a relapse requiring further treatment. The leucocyte count fell, usually within the first two weeks, and the decline for a given daily dose described an exponential function. If the treatment was suspended at a leucocyte count of 11,000 per cubic millimetre or more, the count tended to rise shortly afterwards; but if the count was less than this figure, the effects of treatment were more lasting. In four instances the decline continued to temporarily leucopenic levels. One patient developed pancytopenia. Immature granulocytes disappeared from the blood before the total leucocyte count reached 10,000 per cubic millimetre. Lymphocytes were not greatly affected

<sup>1</sup> *J. Forensic Med.*, July-September, 1954.

<sup>2</sup> *J. Neurol., Neurosurg. & Psychiat.*, November, 1954.

<sup>3</sup> *Northwest Med.*, December, 1954.

<sup>1</sup> *Blood*, September, 1955.

<sup>2</sup> *Arch. Int. Med.*, October, 1955.



by the treatment, and in four cases large numbers of basophilic cells persisted in the blood. Anæmia was found to be ameliorated by successive treatments, and in four cases the number of platelets fell to thrombocytopenic levels. In each of the 15 patients with splenomegaly there was a reduction in the size of the spleen, and in several, although it had previously been massive, the organ became palpable after therapy. Retreatment was instituted when the leucocyte count had increased to the range of from 50,000 to 100,000 cells per cubic millimetre, despite the fact that the patients remained symptom free. Remissions lasted for three months to a year in those cases in which the leucocyte count was reduced by therapy to below 11,000 per cubic millimetre. Treatment tended to be more effective with succeeding courses, and in no case was there either a failure of response or the transformation to the "acute myeloblastic phase". Two deaths were attributable to the treatment; one patient died twelve months after the development of pancytopenia, and the other, with thrombocytopenia, died of cerebral hæmorrhage. It was considered that these complications, in patients in whom they developed, were the results of excessive dosage.

It is perhaps too early to suggest that "Myleran" is the answer to the problem of myelocytic leuchæmia. Little is known of the actual mode of action, and there has yet been no adequate time for trial over several years. Reports may indicate that a maintenance dose of "Myleran" will be sufficient to prevent relapses, rather than treating each one as it appears. Certainly the possibilities are encouraging, especially if knowledge of the mode of action of "Myleran" should lead to other compounds with a similar property of inhibiting abnormal cellular proliferation in the other leuchæmias.

#### FLUORIDATION AND THE TEETH.

THE fluoridation of water supplies as a means of preventing dental caries is now a procedure widely adopted by the authorities in the United States and Canada in charge of the public health of large communities. From time to time a wave of agitation is moved against fluoridation, and with mistaken enthusiasm unpleasant predictions are made of the alarming ill-effects which fluoridation may produce. In fact, natural fluoridation of water supplies exists in many parts of the world; the natives of such regions suffer no ill effects, and it was the observation of the relatively low incidence of dental caries in such people that led to the identification of fluorine as a valuable preventive agent. It may be as well to look back over the whole question of prevention of dental decay, and the particular role that fluoridation can play.

It is a curious reflection on the modern way of living that, despite the almost morbid public interest in disease and the constant struggle to maintain the appearance and vigour of youth, so little should be done to preserve the teeth. A strong, healthy dentition is probably one of the most valuable assets to health and happiness; yet many parents never attempt to instil into their children the habit of cleaning the teeth. At the best, dental hygiene is regarded as a hurried anti-climax before climbing into bed, and the lack of parental example is frequently followed by complete relapse of even daily cleaning when adolescence is reached. A recent editorial article<sup>1</sup> suggested that probably less than one-third of the United Kingdom population uses a dentifrice regularly. The same editorial had also some comment to make on the commercial attitude of the manufacturers of toothpaste. As a whole, this industry is concerned, not with the efficiency of the product in maintaining oral hygiene, but with the ability to make money. Toothpaste advertisements primarily did good by reminding the public of the necessity for regular dental hygiene. Now the advertisements are becoming more spectacular and less accurate, and extravagant claims have been made

for the protective powers of various products without evidence that this is so. The British Dental Association has issued a statement to the effect that they "recognize that a dentifrice is of considerable value in the cleaning of the teeth and gums, but do not accept as proved on the evidence at present available any claim that a dentifrice can actively prevent dental disease otherwise than by virtue of its function as a cleansing agent".

It is now generally accepted that mere regular cleaning may not be so important as a proper diet in the prevention of dental diseases. Communities with a diet low in white flour products appear to possess better teeth. More recently the sweet carbohydrates have been more heavily implicated as agents associated with dental caries, and the widespread practice of stuffing the troublesome childish mouth with a sticky mass of toffee has come under the heavy fire of criticism. Sugar products form a large feature in the diet of children, especially as rewards for acceptable behaviour. Sugar, from almost every point of view, is a poor foodstuff; but once the sugar habit is established, it is almost impossible to break it by voluntary means. In a comprehensive survey of the effect of diet on the teeth, F. W. Clements<sup>2</sup> quoted astonishing figures for the extent of sugar consumption and of its role in the average Australian diet. He referred to the relative absence of dental caries in children who had been fed on a diet containing no refined sugar. These children had far less dental disease than was reported by G. J. Parfitt<sup>3</sup> in a study of 3000 English children, of whom only 1% were found to be caries-free at the age of nine years.

The importance of dental hygiene, and the association of dental caries with sugar products, have been suspected for many years. As a result, the most conscientious parents have instilled some dental hygiene habits into their children, and have restricted their sweet intake and provided a more rationally balanced diet. Unfortunately, voluntary measures are availed of only by the good parents. Children, whatever unfortunate genes they may have acquired, are the victims of poor parenthood. Education may eventually produce generally good parents; but in the meantime it is part of the responsibility of society to ensure that the children receive a fair start in life before they are sufficiently old to care for their own physical assets. Dental hygiene, even in schools, may be on a voluntary basis. What is needed is a procedure which can be generally and cheaply applied, which is harmless, free, and in its application devoid of physical effort. It is in this field that fluoridation of water supplies may play a not inconsiderable part.

However, the question of fluoridation has not received universal support even from medical authority. In a series of three papers F. B. Exner<sup>4</sup> has stated his objections and detailed the contraindications to the use of the fluoridation of domestic water supply, in the prevention of dental caries. He suggests that fluoride, even if accepted as safe and effective, should not be added to the domestic water supply, as the control of dosage is impossible. Fluoride can be given more cheaply by controllable and voluntary methods. The good effects of fluorine are on cells of the tooth buds and on surrounding structures and not on the enamel of the erupted tooth. The health of the tooth depends on the health of these surrounding structures, and these may be adversely affected by fluorine. The most familiar effect of chronic fluorine intoxication is that of mottled enamel or dental fluorosis. Continuation of the toxic fluoridation may result in calcification in the dental pulp, and Exner suggests that the teeth may develop crookedly because of stunting in the growth of the jaws with particular malposition of the third molars. Also, dental caries, once established in mottled teeth, may advance more rapidly, and repair may be more difficult. Calcium metabolism may also be upset by the presence of fluorine. Exner is very critical of all the good reports which have been made of the effects of fluorine and of reasons for and methods of its use. He suggests that the

<sup>1</sup> M. J. AUSTRALIA, February 26, 1955.

<sup>2</sup> Brit. Dent. J., December 20, 1955.

<sup>3</sup> Northwest Med., September, October and November, 1955.

<sup>4</sup> Brit. Dent. J., January 3, 1956.



apparent good effects of natural fluoridation are really due to the presence of natural high calcium concentrations. The methods of making statistical comparisons and the criteria of estimating the individual amounts of dental caries are heavily criticized. In his final paper Exner imputes such motives to the enthusiasts of fluoridation that the whole value of his scientific criticism is cast in doubt. He sees in the recommendations and proposals of others the menace of the totalitarian "Führer", the empire building of the bureaucrat and the pressure groups of the chemical industry. His personal attacks leave no doubt about the strength of his feelings on this point. His sweeping criticisms will no doubt be widely quoted by the antagonists of fluoridation; but though they may mislead some public groups, they are likely to disturb responsible scientific thought very little.

J. M. Mather<sup>2</sup> has reviewed the importance of fluoridation in the dental health of the community. He states that it was originally found that among children drinking water from supplies containing one part per million of fluoride, the rate of dental caries was about 60% less than that of children drinking fluoride-free water. Fluoridation of water supplies in America was begun in 1945. A detailed study of three Ontario cities has been made over the ten years since the introduction of fluoridation. In one, the water supply contained sufficient fluoride. Fluoride, one part per million, was added to the water supply of one of the two fluoride-free cities, which in 1948 had similar rates of caries in their child population. After the introduction of fluoride, the incidence of dental caries in the treated city gradually fell until it was about half that of the untreated, fluoride-free city. By 1954, for the six to eight year age group, the incidence of dental caries in the city with the treated water supply was 69% less than that in the city with a fluoride-free supply, and was the same as that in the city with the natural fluoride intake. The incidence of dental caries also fell significantly in older children who had not received fluoride throughout their lives. No ill effects have been noted at any time. Longer surveys have failed to substantiate the allegations that fluoridation is in any way harmful.

E. R. Schlesinger, D. E. Overton and H. C. Chase<sup>3</sup> have made a detailed survey of the urinary excretion of albumin and formed elements from two groups of twelve year old boys, 100 from a city whose water supply has been fluoridated for eight years and 100 from a city with no fluorine in the drinking water. The averages of albumin levels and of the numbers of red blood cells and casts were found to be rather lower in those children receiving the fluoridated water.

The report of the United Kingdom mission which has investigated fluoridation of water supplies in North America has advised selected trial of the measure. As a result, four British towns<sup>4</sup> have agreed to cooperate by the fluoridation of their domestic water supplies.

The method of the fluoridation needs very careful supervision. In the case of each particular water supply the presence of natural fluoride will determine the amount of fluorine to be added in order to bring the concentration to the accepted level of one part per million. Provision must be made for the accurate mixing of the added material. The determination of fluoride concentration would form a routine part of water testing. Closely allied public health measures have set precedence for the general application of fluorine in drinking water. These are the addition of chlorine to the water supply and of iodides to table salt.

The value and safety of this procedure are beyond doubt. At the same time, every effort must be made to educate the public into proper habits of diet and dental hygiene. Fluoridation is not a treatment of dental caries; it prevents the onset of degenerative disease in teeth. Like immunization and even ordinary bodily hygiene, it is a procedure against which there will be the inevitable waves of misguided antagonism. There were probably many who

grumbled and went on complaining when, by the foresight of Dr. Snow, they were denied the cholera-infested waters of the Broad Street pump.

#### POLIOMYELITIS VACCINATION IN CANADA IN 1955.

CANADA was one of the first countries outside the United States of America to adopt a mass vaccination programme against poliomyelitis, and paradoxically has gained a good deal of attention simply because of the quietness and lack of fuss about the programme and the results. The experience gained is, however, of great general interest, although it is too early to draw much in the way of conclusions. The way in which an attempt has been made to evaluate the Canadian experience is described by E. H. Lossing,<sup>1</sup> Chief of the Epidemiology Division of the Canadian Department of National Health and Welfare. It was recognized that any attempt at conducting a study involving the administration of a placebo substance to a control group was impossible. The decision was therefore made to collect information which would allow a simple comparison of attack rates in vaccinated children and unvaccinated children, as similar as possible in age and risk of exposure. For the purposes of the study, because of diagnostic difficulties, only cases of paralytic poliomyelitis were taken into account; and the diagnosis of paralytic poliomyelitis was restricted to cases in which clinical manifestations of poliomyelitis with muscular weakness were present for more than twenty-four hours, as determined by two successive examinations. The observation period of the study was from July 1 to November 30, 1955; July 1 was considered a date by which immunity might be expected to be established in vaccinated children (most vaccinations were given in April, May and June), and November 30 was chosen as an arbitrary termination date, because by then the incidence drops to insignificant levels. The vaccine used was produced by the Connaught Medical Research Laboratories, University of Toronto. The figures quoted by Lossing show that the incidence of poliomyelitis in Canada in 1955 was exceptionally low. The reported incidence rates of poliomyelitis (all forms) for the year was the lowest in ten years, while the incidence of paralytic poliomyelitis was lower than in any year since 1950. In only three of the 11 provinces did the 1955 incidence of paralytic poliomyelitis approach the five-year average. In all provinces where paralytic cases occurred in the vaccinated or unvaccinated group, the attack rate in the unvaccinated group exceeded the attack rate in the vaccinated group of children of comparable ages during the period of observation. In provinces where the 1955 incidence of paralytic poliomyelitis was low in relation to the five-year average, these differences, although observed, were not statistically significant; but in the three provinces where the 1955 incidence of paralytic poliomyelitis more nearly approached the five-year average, these observed differences in the vaccinated and unvaccinated groups have statistical significance, if it is assumed that the vaccinated and unvaccinated populations were similar in other respects. It is therefore concluded from the study that a protective effect from the vaccine might be inferred in areas where the 1955 incidence was low, and was demonstrated where the 1955 incidence more nearly approached the five-year average. These conclusions are drawn cautiously and with certain reservations in the light of the many variable and unpredictable factors known to exist. At the least, however, we may say that a beneficial result from the vaccination programme is consistent with the facts.

On another important point, the safety of the vaccine, Lossing's comments are interesting. He writes that it is an understatement to say that considerable concern has been felt and expressed about the safety of the vaccine in Canada and elsewhere. If the vaccine contained live virus, it would be reasonable to expect that either clinically recognizable poliomyelitis would be observed among vaccinated children, or if live virus were introduced with

<sup>1</sup> *Canad. J. Pub. Health*, January, 1956.

<sup>2</sup> *J.A.M.A.*, January 7, 1956.

<sup>3</sup> "Facts and Fancies on Fluoridation", *Brit. Dent. J.*, January 17, 1956.

<sup>4</sup> *Canad. J. Pub. Health*, March, 1956.

resulting subclinical infection or a "carrier" state in the vaccinated child, clinically recognizable poliomyelitis might be expected to occur in the family contacts of these vaccinated children, within a double incubation period after vaccination. However, no case of poliomyelitis has been reported in vaccinated children in which the date of onset was within one month of vaccination. Similarly, inquiries have revealed no significant occurrence of recognizable poliomyelitis in contacts of asymptomatic vaccinated children within thirty days of vaccination. It is therefore concluded that the vaccine used in Canada in 1955 was safe.

#### CORTISONE AND SALICYLATE IN RHEUMATOID ARTHRITIS.

THREE large trials of the effect of cortisone on rheumatoid arthritis have been reported by the British Medical Research Council, the Nuffield Foundation, and the Joint Anglo-American Working Party on Rheumatic Fever. These trials were concerned with the efficiency of cortisone in comparison with older therapeutic agents in the treatment of rheumatic fever and of early cases of rheumatoid arthritis, and as an adjuvant to manipulation. None of these trials demonstrated that cortisone had any striking overall advantage as compared with salicylate. None the less, the impression remained that in a number of patients with rheumatoid arthritis the effects of cortisone were rapid and favourable. Accordingly a further trial was conducted by the Empire Rheumatism Council Research Subcommittee, to test the effectiveness of cortisone in patients with long-standing rheumatoid arthritis. The points of inquiry in this survey were the clinical and functional efficiency of aspirin and cortisone over periods of up to three years, the radiological appearances in the two groups at yearly intervals, the rate of treatment intolerance, and the incidence of complications.

One hundred patients were selected, with purely rheumatoid polyarthritides, with no grossly irreversible changes in the joints, with no other complicating diseases, and between the ages of seventeen and sixty years. Patients were distributed among nine treatment centres. To half the patients cortisone was administered in a dosage of 25 milligrammes by mouth every eight hours until the maximum effect had been produced, when the dosage was reduced or increased to the minimum required to keep the patient symptom-free. Patients needing less than 25 milligrammes per day or more than 100 milligrammes per day were rejected from the trial. To the other patients in this trial acetyl salicylic acid was given in five daily doses of four grammes per day until the maximum effect had been produced, and then the dosage was reduced to the minimum necessary, between one and eight grammes per day. The report<sup>1</sup> has now been made of the first year of the trials. Of the patients in the trial, 38 completed the first year on cortisone and 39 on aspirin. The patients were fully examined regularly, and notes were made of the patients' subjective opinions, the state of the joints, the haemoglobin concentration, the erythrocyte sedimentation rate and any complications. In addition, patients were assessed into four grades of employability and four grades of functional capacity. Of the joints, 68 were regularly examined for involvement in the disease, and radiological assessment of two affected joints was made at six-monthly intervals. Five patients proved to be intolerant of aspirin, and four patients were taken off cortisone therapy because of complications. Equal numbers of patients withdrew from the two groups because of lack of benefit from the treatment. Mild side-effects and complications of treatment occurred in 18 patients receiving cortisone and in 17 patients receiving salicylates. The mean duration of symptoms was about seven years in both groups, and the groups compared well together in regard to age, sex, clinical condition, employment status, and functional capacity. In neither group was there any real difference in the number of joints affected at any time during the first year. The erythrocyte sedimen-

tation rates fell gradually in both groups after the usual temporary depression of the rate in those patients receiving cortisone. Both groups had a low average haemoglobin level of 12.5 grammes *per centum*, and the only significant change in the whole trial was a rise of this level to 13.3 grammes *per centum* average in those patients receiving cortisone. No significant differences were observed in routine radiological assessment of the joints of the wrists and hands in each of the patients. The assessment of improvement in the general condition was difficult, but on the combined improvement in employment status, objective function and subjective function, it was found that five patients receiving cortisone and seven patients receiving aspirin had improved over the first year of the survey. It is concluded in this report of the Empire Rheumatism Council Research Subcommittee that there is no significant difference after one year's treatment with either cortisone or aspirin in patients suffering from chronic rheumatoid arthritis.

#### SURGICAL WOUNDS WITHOUT DRESSINGS.

FROM time to time surgeons have reported that they have left their surgical wounds without dressings either as a routine or on certain occasions, with satisfactory results. Certain advantages are, of course, immediately apparent—namely, increased convenience to the patient and a considerable saving in materials and time taken to change dressings. The objections seem not to be formidable. When C. J. Helfetz *et alii*<sup>1</sup> reported on their experimental and clinical experience of this practice in 1953, they were quite strong in their praise of its value in the right circumstances, but offered a list of occasions when it was not advisable, including when local anaesthesia had been used. In a recent series reported by L. T. Palumbo, P. J. Monnig<sup>2</sup> and D. E. Wilkinson,<sup>3</sup> there is no mention of the use of local anaesthesia, but otherwise their experience suggests that there is little in the way of contraindication to the abandonment of post-operative dressings. Palumbo and his colleagues point out that it has been shown experimentally that fresh clean wound margins are sealed in a matter of hours after being sutured, that the serum agglutination of the wound margins provides a favourable barrier to any outside contaminant, and that a wound of this type exposed to the air is unlikely to become moist and so become a nidus for the debris and bacteria which can accumulate under a surgical dressing. They report a controlled study of 222 clean major surgical wounds of the thorax and abdomen of 211 patients. One half were treated by the routine method of applying clean sterile surgical dressings, which were changed as necessary and in most cases removed by the eighth day; the others were uncovered early, some within six hours and all within forty-eight hours. The complications were not serious in either group and were practically identical in the two groups. On the other hand, the wounds without dressings appeared to heal more rapidly and with less local inflammation. It is interesting to note that the patients offered no objection to the scheme, and they (not to mention their visitors) were eager to watch their wounds during the healing process. The wounds were not irritated by bed covers or pyjamas. Inguinal hernioplasty wounds healed as readily, without complications, as wounds in other areas of the abdomen. Altogether, from the point of view of clinical progress, the uncovered wounds did at least as well as those with surgical dressings. Practical advantages of the no-dressing technique were the convenience, the saving in cost of surgical dressings, the saving of time of professional staff in wound care, the elimination of cumbersome dressings and the absence of adhesive tape irritation. It all sounds very attractive, and certainly further clinical trial of the method is warranted. It is to be hoped that it will prove satisfactory in long-term experience, but many surgeons will feel cautious about it, especially in these days when wound infection with antibiotic-resistant organisms is a major problem in some hospitals.

<sup>1</sup> Arch. Surg., November, 1953.

<sup>2</sup> J.A.M.A., February 18, 1956.

<sup>3</sup> Ann. Rheumat. Dis., December, 1955.



## Abstracts from Medical Literature.

### OPHTHALMOLOGY.

#### Hereditary Eye Disease.

P. T. MANCHESTER (*Am. J. Ophthalm.*, September, 1955) writes an instructive paper on advising patients with hereditary eye disease. He elucidates the problem by the use of tables showing how to determine the mode of inheritance, and giving the answer to the question: "What are the chances my next child will develop the disease?" Finally he indicates all the important hereditary diseases. The paper is intended to help medical practitioners to answer problems in heredity which confront them from time to time.

#### Fuchs's Syndrome of Heterochromic Cyclitis.

S. J. KIMURA *et alii* (*Arch. Ophthalm.*, August, 1955) review the syndrome of heterochromic cyclitis. They state that the disease occurs most frequently in the third or fourth decades of life with an insidious onset and a very long course. In a fully developed case there are heterochromia, cyclitis and cataract. In addition, peripheral choroiditis may be observed. Heterochromia is not a consistent sign, and the disease may be identified in its absence. The aetiology of the disease is obscure, and there is no known treatment. Cortisone has no effect. The cataracts when they develop are easily removed, and the eye responds well in spite of the presence of cyclitis.

#### Hypotony After Cataract Extraction.

J. H. DUNNINGTON (*Brit. J. Ophthalm.*, January, 1956) divides cases of hypotony occurring after removal of a cataract into two types. In one there is a shallow or absent anterior chamber with choroidal detachment. The cornea is clear at first, but becomes hazy if the condition persists. The anterior chamber is shallow or absent. The vitreous is ballooned forward through the pupil and often touches the cornea. Extensive choroidal detachment is always present. The condition occurs spontaneously in the second week after operation and may be preceded by hyphema. If the condition persists for four or five days, glaucoma is practically certain to occur. In the second type of hypotony there is delayed fistulization without loss of the anterior chamber. The usual history is one of an uncomplicated operation with a normal post-operative course. After a few weeks or months the eye becomes uncomfortable with episodes of blurred vision and lachrymation. Between attacks the vision is normal, the cornea is clear and the anterior chamber is of normal depth. The ocular tension is normal or low. The result of a fluorescein test may or may not be positive. As weeks pass the episodes increase in frequency, vision begins to fail, and the globe becomes tender and injected. There is mild ciliary injection, with cells in the anterior chamber, which is of normal depth, fine posterior synechiae and clouding of the anterior part of the vitreous. Papilloedema

and macular oedema may be present. In the first type spontaneous reformation of the anterior chamber is the rule. If the condition persists for four or five days, any corneo-scleral sutures present should be removed, and air should be injected into the anterior chamber together with the release of subchoroidal fluid. In the second type surgical treatment to close the fistula is required.

#### Uveitis.

G. BENNETT (*Brit. J. Ophthalm.*, December, 1955) has devised a classification of uveitis after studying 332 cases of primary uveitis. The disease was unilateral in 64% of the patients. The sexes were equally affected except when the aetiological factor was one of rheumatoid arthritis or ankylosing spondylitis. The study confirmed the fact that uveitis is rare in childhood; there were only seven cases in patients under fifteen years of age. The erythrocyte sedimentation rate was higher in cases of uveitis associated with systemic disease than in those with purely ocular conditions. The investigation and treatment of focal sepsis appeared to have no effect on the relapse rate. Raised ocular tension occurred more frequently in cases due to local virus infection. Old tuberculin proved to be useless in treatment. Cortisone might be beneficial in cases associated with collagen disease, and dangerous in cases due to virus disease. The author comments that the investigation indicated the worthlessness of much expensive clinical investigation of patients and of their therapeutics, and urges that greater attention be paid to old fashioned history-taking and to clinical study of the patient as a whole.

#### Flattened Anterior Chamber.

J. BELLOWES *et alii* (*Arch. Ophthalm.*, August, 1955) discuss the problem of flattened anterior chamber occurring after cataract operations. On the basis of animal experiments and of clinical material they offer a logical explanation of the persistence of a flattened anterior chamber after surgical removal of a cataract. A primary consideration in the prophylaxis of this complication is to secure a water-tight wound. A complete iridectomy would seem to be of definite prophylactic value. In the absence of a defect in the wound a flattened anterior chamber should be treated by mydriatics; and if this does not improve matters, acetazolamide ("Diamox") may be tried. If the anterior chamber has not reformed by the fourth or fifth day after operation, it is advisable to inject an air bubble into the anterior chamber; if this fails, then posterior sclerotomy should be performed together with the injection of air into the anterior chamber. If this last procedure fails, then consideration should be given to incising the anterior face of the vitreous.

#### Peripheral Iris Crypts.

A. POSNER (*Am. J. Ophthalm.*, October, 1955) discusses the mechanism of narrow angle glaucoma and explains why some eyes with shallow angles never develop glaucoma. He has found that eyes with well developed peripheral iris crypts are not subject to elevations of tension.

The crypt acts like an iridectomy to prevent the closure of the angle by anterior suction on the iris, even though it does not extend through the entire thickness of the iris. A single crypt can be as effective as a large number of them in preventing acute glaucoma.

#### Glaucoma Treated with "Diamox".

S. M. DRANCE (*Brit. J. Ophthalm.*, November, 1955) reports on the use of "Diamox" in 20 patients with glaucoma. In cases of congestive glaucoma 500 milligrammes of "Diamox" were given by mouth and repeated after six hours if necessary. In all other cases 250 milligrammes of "Diamox" were given by mouth every six hours with 30 grains of sodium bicarbonate. When the tension was controlled, the dosage was reduced to 125 milligrammes given six-hourly. All patients complained of tingling in the fingers and some of dyspepsia while taking the drug. The drug seemed to be most valuable for secondary and congestive glaucoma.

#### The Management of Monocular Cataracts.

E. L. GOAR (*Arch. Ophthalm.*, July, 1955) considers that a patient with a monocular cataract should have that cataract removed. Failure to do this may lead to exotropia, hypermaturity and secondary glaucoma. After the removal of the cataract, the author recommends that a contact lens should be ordered for the aphakic eye. In his experience these patients do not experience discomfort.

#### The Ocular Significance of Intracranial Calcium Deposits.

J. E. ALFANO and H. WHITE (*Arch. Ophthalm.*, July, 1955) discuss ocular diseases which are associated with intracranial calcification. These include toxoplasmosis, Sturge-Weber disease, tuberous sclerosis, congenital vascular malformations, intracranial tumours and intracranial arterial aneurysm. In skiagrams of the skull it is to be remembered that radiological calcification may be seen in the cornea, in the orbit in retinoblastoma, and in the intracranial portion of the optic canal.

#### Goniotomy.

G. S. TYNER and E. J. SWETS (*Arch. Ophthalm.*, July, 1955) report on the treatment of glaucoma by the operation of goniotomy. Thirteen eyes were treated, and the tension was controlled in nine, including two eyes with juvenile glaucoma, two with aniridia and four with congenital glaucoma. Repeated punctures were necessary in most cases, and no serious complications were encountered. The authors indicate that none of their cases have been reviewed long enough to give final evaluation of the procedure.

#### Pathogenesis of Congenital Glaucoma.

O. BARKAN (*Am. J. Ophthalm.*, July, 1955) traces the development of the normal angle of the anterior chamber from infancy to adulthood by microgonioscopic examination of more than 40 normal eyes. He describes in detail the normal angle



and its changes from infancy to adult life. He then describes the changes in the angle and in Schlemm's canal in cases of congenital glaucoma.

### The Morgagnian Cataract.

W. C. CAOCAMISE (*Am. J. Ophthalm.*, July, 1955) discusses features of morgagnian cataract. He classifies senile cataract as incipient, immature, intumescent, mature and hypermature, which may be shrunken or morgagnian. The morgagnian cataract presents a homogeneous, milky, white surface. The brown sclerotic nucleus lies in a capsular sac of milky white, liquefied cortex. In general all morgagnian cataracts should be removed, as an eye with such a cataract is liable to the development of subluxation of the lens, glaucoma and uveitis. For removal the author recommends an intracapsular technique, but when he removed the lens by the extracapsular technique the results were good. He recommends the Smith Indian operation or the use of an erisophake for extraction.

### Etiology and Therapy of Retinal Vascular Occlusions.

B. ANDERSON AND W. VALLOTTON (*Arch. Ophthalm.*, July, 1955) find that the great majority of venous or arterial occlusions are associated with arteriosclerosis and hypertension. Whatever the cause of the obstruction the immediate aim of the treatment is to increase the calibre of the vessel which has become constricted. In arterial occlusions the authors recommend the retrobulbar injection of a 2% solution of procaine together with 250 units of hyaluronidase. In addition 10 to 25 milligrammes of "Priscoline" are also given with the retrobulbar injection. They also recommend injection of the stellate ganglion. In arterial and venous occlusions anticoagulant therapy is recommended. The authors continue anticoagulant therapy for thirty to sixty days. The results of treatment in the author's series were disappointing.

### Intravenous Treatment of Optic Neuritis.

P. KAZDAN AND R. KENNEDY (*Arch. Ophthalm.*, May, 1955) report on the use of both ACTH and a solution of killed typhoid bacilli given by the intravenous route in the treatment of optic neuritis. Eleven patients were treated with ACTH and 11 with intravenously administered killed typhoid bacilli. Comparison of the results obtained from these two methods of treatment showed no superiority of one method over the other, especially as regards visual acuity.

### Cyclodiathermy.

H. G. SCHREIE *et alii* (*Arch. Ophthalm.*, June, 1955) present the results obtained in 38 eyes treated by cyclodiathermy. The authors use a 1.0 to 1.5 millimetre electrode applied through the conjunctiva six millimetres behind the limbus at eight to ten points evenly spaced to cover slightly more than half the globe. The tension was controlled in 15 eyes without miotics and in six more that had miotics given post-operatively. Results

were good for a few months, but tension tended to rise again about one year after operation. The authors recommend the operation when more conventional surgery has failed, or when ordinary surgical methods are considered to be hazardous.

### OTO-RHINO-LARYNGOLOGY.

#### Stapes Mobilization in Otosclerosis.

Y. MUERMAN AND O. MUERMAN (*Arch. Otolaryng.*, August, 1955) operated on 63 patients, using the Rosen technique, in Helsinki, Finland. There were 46 women and 17 men. According to hearing acuity, these were capable of classification into three groups. Classification was according to standards of bone conduction and air conduction, much as is followed in selecting cases for fenestration operations. In the first grade there were 40 cases. Of these, 19 obtained a good improvement (20 decibels or more), five had slight improvement (15 to 20 decibels), and in 16 the hearing was unchanged. The proportion of good results was less in the second and third grade of cases. In none of the patients did an impairment of hearing take place when the improvement had reached a maximum. The degree of stapes fixation seemed to have some bearing on the results obtained. In two-thirds there was distinct improvement when the stapes was easily mobilized. When it was firmly fixed, hearing improved in only half the cases. A tear of the tympanic membrane was found to impair the hearing improvement considerably. The original incision must be placed at least five millimetres lateral to the drum margin in order to avoid a persisting open slit at the border of the tympanic membrane. The chief cause of failure is rupture of the crura of the stapes, a happening which can be avoided only by the utmost caution and the employment of repeated even pressure and non-jerky movements. A binocular Zeiss microscope giving six to 16 magnifications was found to be of valuable assistance. The operation is concluded to be a worthwhile procedure. The improvements attained in many cases exceeded those produced by fenestration. The procedure is a much smaller surgical task. If it is unsuccessful, a fenestration operation can still be performed.

#### Papilloma of the Larynx in Children.

H. W. KOHLMOOS (*Arch. Otolaryng.*, September, 1955) hopes from a review of the literature covering aetiology, various methods of treatment and careful study of a number of cases of papilloma of the larynx in children, that he may be able to determine the optimum treatment for this disorder. The lesions in children are usually multiple and tend to recur, while in adults solitary lesions are more common. The aetiology remains unproven. It has been suggested that the lesions, as occurring in children, are of connective tissue origin rather than a papillary hypertrophy of epithelial origin. Several authorities have concluded that these papillomata are an inflammatory reaction probably due to a filtrable virus. Some

promising results with the use of oestrogenic hormones stimulated interest in endocrine aetiological factors, but conclusive evidence has not been presented. The self-limited nature of the disorder and the usual clearance at puberty are points stressed in favour of endocrine influences. The author concludes that laryngeal papillomata of children must still be classified as true neoplasms. Papilloma of the larynx is not uncommon. In most reports the incidence is highest in the one to four years age group, and is approximately equal in males and females. There is no evidence of unusual racial or geographical distribution. Hoarseness is the symptom common to all cases. Respiratory obstruction with wheeze, noisy breathing, sternal retraction and finally acute respiratory distress develops as the glottis fills with tumour. Tracheotomy is often necessary for this obstruction and may be needed for several years. In a number of instances lower respiratory obstruction due to tracheal implants has called for repeated bronchoscopy through the tracheotomy. In mild cases removal of the tumour once yearly for two or three years has been sufficient, whilst in others more than 100 procedures have been necessary. Spontaneous recovery with complete disappearance of the tumour masses before puberty is almost universal. Papilloma of the larynx is easily diagnosed by inspection and after biopsy. Prior to such procedures a variety of disorders causing hoarseness and respiratory obstruction must be considered. A great variety of treatments have been described. None are as yet universally successful. Long courses of arsenicals, potassium iodide and calcined magnesias have been advocated, but no persistent cures have been reported. The local applications of oestrogenic hormones were optimistically reported. Also local applications of podophyllum resin were thought to lessen the tumour growth and increase the ease of removal. External irradiation or radium applications were first held to give promising results. The use of these agents in children has, however, been condemned as likely to cause scarring, stenosis and poor development of the larynx, while persisting growth of the papillomata was noted. The use of antibiotics has been advocated, and in some cases has produced favourable results. However, it has not been wholly successful, although it may be an adjunct to surgical removal. The consensus of many authorities is that careful removal of the tumour masses to ensure an adequate airway, with tracheotomy where required, is the optimum treatment. The disorder is self-limited and will clear with age. Operative procedures should not be so radical as to injure normal structures, lest stenosis or cartilage damage may result, possibly with a permanently damaged voice. Fifteen cases from the author's personal experience are reviewed, and the findings and various measures employed in treatment support the summary of evidence from the literature. It seemed that the patients with sessile multiple lesions required more frequent treatments, and more often needed tracheotomy, than those in whom the lesions were solitary and not diffuse in nature.

## Clinico-Pathological Conferences.

### A CONFERENCE AT SYDNEY HOSPITAL.

A CLINICO-PATHOLOGICAL CONFERENCE was held at Sydney Hospital on Tuesday, January 17, 1956, the medical superintendent, Dr. NORMAN H. ROSE, in the chair. The principal speaker was Dr. T. E. WILSON, an honorary assistant surgeon of the hospital.

#### Clinical History.

The patient was a sixty-year-old male, a labourer by occupation, who was admitted to hospital with a history of an attack of tenesmus and frequent passage of blood and mucus per rectum three months prior to admission. This subsided, but at the time of admission he still had some tenesmus and passed a good deal of wind and some mucus occasionally but no blood. His appetite was good, but he had lost twelve pounds in weight over the previous three months. There was no abdominal pain, nausea, vomiting or other relevant symptoms. Previously he had had an appendicectomy thirty years before, "colitis" ten years before (with blood and mucus in the stools), removal of a rodent ulcer on the forehead four years before and bronchitis one year before. He smoked half an ounce of tobacco weekly and drank "moderately".

Physical examination of the patient showed that the tongue was moist and furred. The abdomen moved evenly with respiration and was without tenderness, guarding or rigidity. The liver and spleen were not palpable, but there was a non-tender mass in the mid-line of the abdomen just above the umbilicus, which moved with respiration. No other abnormality was detected in any other system. The urine was free of albumin and sugar. A barium enema X-ray examination performed prior to admission showed a constant area of constriction in the transverse colon, and a provisional diagnosis of carcinoma of the colon was made.

Three days after the patient's admission to hospital, laparotomy was performed through a left paramedian incision. A stricture of the transverse colon two inches long was found together with some enlarged lymph nodes in the transverse mesocolon. This was thought to be either neoplastic or inflammatory. Twelve inches of transverse colon together with its mesentery were resected, and an end-to-end anastomosis was performed. The loop of bowel containing the anastomosis was brought exterior to the peritoneum and muscle, and the overlying skin was left open in the central part.

The immediate post-operative course was uneventful, but on the fourth day faeces commenced to drain freely from the wound. Faeces continued to discharge freely, and the surrounding skin became excoriated. The abdomen was soft and not distended, but nothing was passed per rectum. By the end of the fourth week there was gangrenous skin four inches in diameter at the centre of the wound, and a diagnosis of symbiotic gangrene was made. A week later the gangrenous skin and its underlying adipose tissue were excised.

At first the excised area remained clear and granulated well, but later it appeared inflamed (not responding to local injections of penicillin), and within a month gangrene had reappeared. Faecal discharge had been continuous.

He then commenced to vomit and have colicky abdominal pain, and no faeces were passed either through the fistula or per rectum.

The true diagnosis of his condition was made at this juncture, but in spite of treatment he died five days later, eight weeks after his entry to hospital. His temperature had risen each evening to 100° or 101° F. from the tenth post-operative day, when excoriation of the skin was first noted, till his death.

The speaker today is invited to attend the patient as a consultant four or five days before he died and to discuss diagnosis and management.

#### Results of Special Tests.

**Resected Bowel.**—Macroscopic specimen: Portion of the colon measuring 12 centimetres in length was received. The bowel was constricted five centimetres from one end. There was only a small inconspicuous area of ulceration with no macroscopic evidence of extension into the adjacent adipose tissue. Microscopic examination: The ulcer base was formed by granulation tissue containing many plasma cells, lymphocytes and polymorphonuclear cells. There was no evidence of carcinoma.

**Blood.**—On the patient's admission to hospital the haemoglobin value was 15.9 grammes per centum. Two weeks before his death the haemoglobin value was 9.79 grammes per centum (normocytic, normochromic anaemia), the white cell count was 13,700 per cubic millimetre (neutrophils cells 47%, shifted to left, eosinophile cells 4%, lymphocytes 23%, monocytes 12%).

**Gangrenous Area.**—Direct smear examination showed fairly numerous pus cells, Gram-negative bacilli and occasional Gram-positive cocci. Culture (aerobic and anaerobic) revealed *Bacillus proteus*.

#### Clinical Discussion.

DR. T. E. WILSON: This patient presented with a story typical of a carcinoma of the colon, although there was a mass present which proved to be an inflammatory mass. The differentiation of this from a neoplastic mass was not very important prior to the operation, for in either case resection of the lesion was indicated to relieve the obstruction. Even if the surgeon had been able to determine prior to the operation the true nature of the mass, the treatment would have been much the same. There are no details in the history we have been given today of the pre-operative preparation; for example, whether the patient had chemotherapy. When resection of large bowel is indicated, the choice of technique—a one-stage resection, or a preliminary colostomy with resection later, or a Paul-Mikulicz operation—has to be considered in the light of the pre-operative preparation. Usually we know the patient has been prepared with phthalylsulphathiazole, streptomycin *et cetera*, and a one-stage resection and anastomosis is performed as long as there is no infection, obstruction or gross adhesions. Normally a man like this would come to operation after some such preparation, but if he had not been properly prepared a two-stage procedure would have been indicated.

In this case, after the resection and anastomosis was performed, the loop of bowel was brought into a subcutaneous position, presumably because the surgeon was in doubt about his anastomosis or the viability of the bowel. However, I would not recommend the procedure of bringing the loop of bowel out into that position. If there is any doubt about the viability of the bowel ends, further resection is indicated. The development of a faecal fistula showed that the surgeon was right in his doubts, and this proves that more of the bowel should have been resected.

The commonest serious complication after a colostomy is the adherence of a loop of small bowel to the side of the colostomy and then kinking with obstruction. In this case, although the patient did not have a man-made colostomy, much the same effect was produced with a loop of bowel going through the peritoneum. I feel quite sure that the terminal illness of this patient, with its vomiting, abdominal pain and absolute constipation, indicated that a loop of small bowel had become adherent and kinked where the transverse colon had been brought up to the surface.

I have been asked to discuss the patient from the viewpoint of a consultant called in four or five days before he died and to discuss the diagnosis and management at that stage. The inflammation of the skin and subcutaneous tissues was probably associated with more intraabdominal adhesions and inflammation of the bowel than is normal with a colostomy, but I think that was an accessory factor which did not directly contribute to his death. There was a history some years ago of colitis, but there is no other mention of pathological findings to support this. The patient with chronic ulcerative colitis does not usually get a pericolic mass, but they are liable to suffer a variety of other complications, such as fistulae and strictures. The presence of an inflammatory mass sufficient to block the bowel suggests chronic diverticulitis rather than ulcerative colitis. Four or five days before his death it was a question of doing something which would save his life; the diagnosis was then obstruction of the small bowel, and that required relief whatever the cause.

In this patient there may have been a small intraabdominal collection of pus, which would further aid and abet the adhesion of the bowel, but that would have been an incidental finding at the time of the operation. There may also have been a second, although incomplete, obstruction at the time of the initial operation. I do not think it would have been overlooked; but even if it had, it would probably have produced signs and symptoms earlier. Even so, whatever the cause, laparotomy was indicated to relieve the obstruction; and I presume that when it is stated that "in spite of treatment he died", it means that laparotomy was included in the treatment. The incision would have been placed as near as possible to the colostomy, though there would have been the difficulty of having to go wide of the symbiotic gangrene. The operation would have required the



services of an expert anaesthetist together with the minimum amount of operative trauma. The mortality rate of intestinal obstruction used to be 40% to 60%, and it is still fairly high. The correct operation for intestinal obstruction is the minimum procedure which would relieve the obstruction, and this may be just freeing the loop of bowel or, if that is not possible, some internal or external drainage.

The other thing to mention in managing such a case is the question of aspiration and fluid replacement. I will not go fully into this, but there are two points I would like to mention. Firstly, I think aspiration is better when intermittent than continuous. If the tube is passed into the stomach and connected to some continuous suction apparatus, there is always a risk that the patient will not receive the same care and attention, and that blockage of the tube may be overlooked. If the patient has a tube such as a Wangenstein or Jacques tube passed, and this is aspirated quarter-hourly or half-hourly, then I think one gets a better result. The fact that there is a nurse attending the patient at regular intervals is a good thing. I do not think that in a patient like this there is really any place for a Miller-Abbott tube.

I will not discuss the Miller-Abbott tube except to say that though theoretically a wonderful idea it has so many disadvantages that in practice it is rarely indicated. For fluids, emphasis must be on the fact that too little given is not as bad as too much. In a patient like this, who has a long story of many weeks' illness and then develops intestinal obstruction, or even if he did not have a long illness beforehand, one can get into more trouble by giving too much fluid than by giving too little. I am not suggesting keeping the patients dehydrated, but I am pointing out that it is very easy to drown them. The same applies to salt replacement: it is better to give too little than too much. I would suggest, if called to give advice here, that the fluid of choice would be 4% glucose with one-fifth normal saline, normal saline only being used if the urinary chlorides are practically absent. Potassium should be added to the intravenous fluid only if it is definitely indicated by a serum potassium level below 4.1 milliequivalents per litre, if the patient is unable to take it by mouth, and if there is an adequate output of urine. Whether or not other fluids are used is a matter of personal reference, and only under very special circumstances are they indicated.

To summarize, four or five days before the patient died, I would make the diagnosis of intestinal obstruction due to a loop of small bowel becoming adherent to the transverse colon where it was brought to the surface. The management of the case would have been relief of the obstruction in the quickest and easiest possible way with the help of an expert anaesthetist and with special attention to suction and administration of fluids. Like many cases of intestinal obstruction, it is quite likely the patient would have needed blood transfusion.

Was there any other aspect you wanted me to discuss?

DR. NORMAN H. ROSE: One thing which I consider that you have not given enough attention to and I think is important is the abdominal-wall gangrene. We would like your opinion of the cause and treatment.

DR. WILSON: I did not mention that, except in passing, because I do not think it really contributed to the intestinal obstruction which caused his death. I can imagine all the trouble that it must have been to the surgeon, but I think it was a side issue, which no doubt made the patient's life a misery but had little to do with the development of the obstruction. Various treatments of symbiotic gangrene have been described, but none is very effective. The idea of its being a symbiosis between one organism and another I cannot comment on, but from the surgeon's point of view the treatment which should be effective is complete excision. Yet, even following such excision it often reappears. Peroxide paste and the other preparations have some basis for their use, but none is very effective.

DR. ROSE: I think I can remember this patient's demise in hospital, and I can assure those of the audience who are not surgeons that the case holds as much, if not more, interest for the physician.

DR. W. L. CALOV: I was struck by the fact that this man had had an attack of colitis ten years before with blood and mucus in his stools. I assume that that attack of colitis had something to do with the stricture of the transverse colon, and it recurred. Ordinary colitis does not cause stricture of the bowel; it is not a characteristic feature of ordinary ulcerative colitis. But amoebic dysentery will cause stricture. In my experience it is not common for amoebic dysentery to cause tenesmus and a great deal of straining, but, of course, that depends on lesions being sufficiently low in the rectum, and this sometimes does occur in amoebiasis.

I think this patient probably had amoebic dysentery, amoebiasis, and that the strange phenomenon round about the wound was caused by the amoeba together with other organisms, and I think that had a lot to do with his failure to survive. I have no doubt that, as Mr. Wilson said, the man had intestinal obstruction, but I feel sure he had amoebiasis as well. Of course, Mr. Chairman, whenever someone says he feels sure you can be reasonably certain that he is wrong.

DR. ROSE: Yes, I agree with you, Dr. Calov.

DR. W. EVANS: The original diagnosis of carcinoma of the colon was subsequently proved to be wrong, so the nature of the original illness is still in doubt. Dr. Calov has raised the question of amoebiasis; I am not sure that I agree with that, but I have not seen a great deal of this disease. I wonder whether it could have been tuberculosis, but the pathological reports do not favour that. Nevertheless, the attack of colitis some years before must be important, together with its recurrence more recently, and I thought some of the commoner forms of colitis giving rise to constriction should be considered. Following that he suffered at the hands of the surgeon and finally succumbed. I would favour some form of colitis.

DR. J. B. BLACKWELL: I agree with the suggestion of amoebiasis. The original lesion could well have been caused by *Entamoeba histolytica*, which, in addition to the points already raised in its favour, can give rise to a palpable abdominal mass; and following operative procedures in untreated cases of amoebiasis ulceration of the skin may occur.

DR. R. E. J. TEN SELDAM: I would like strongly to support the diagnosis of amoebic dysentery, because this man had colitis with the passage of blood and mucus—though the history does not state which came first; usually in amoebic dysentery the mucus precedes the blood. The presence of skin gangrene, and the constrictive lesion without other lesions in the resected part, are in my opinion not consistent with diverticulitis. Diverticulitis is far commoner in the lower bowel, where stools are less fluid. A clinical point in favour of amoebiasis is the fever, which shows a regular rise in the evening. I have not seen amoebic gangrene following surgery, though I have seen it around the anus and over the scrotum. Of course, in the tropics we were biased, and anyone with doubtful bowel trouble was treated for amoebiasis before anything further was done. But I should not be at all surprised to find that this patient had amoebic dysentery and to hear that amoebae were found in the granulation tissue.

DR. ROSE: Do you suggest, Dr. ten Seldam, that this was an actual amoebic infection of the abdominal wall?

DR. TEN SELDAM: Yes.

DR. ROSE: I will call on Dr. Hirst to give us the pathologist's findings.

#### Pathological Report.

DR. E. HIRST showed slides and gave the pathological findings, of which the following is a summary:

The body was that of a thin old man. On the centre of the anterior abdominal wall there was a large area 23 centimetres by 27 centimetres devoid of skin and superficial fascia. At the edges the skin was heaped up and blackened. This area had been the site of excision of the superficial tissues. There were several shallow pockets of viscid green pus in the neighbouring subcutaneous tissue. A fistula from the centre of the transverse colon opened about one centimetre above the umbilicus.

Only the small intestine and colon showed significant abnormalities. The small intestine was congested and distended with copious faecal fluid to a point 15 centimetres from the ileo-caecal junction. Here two loops of ileum were twisted and adherent to each other and to the abdominal wall just below the fistula. Apart from fistula no abnormality was found in the remaining colon.

#### Operation Specimen from Abdominal Wall.

An approximately circular piece of skin and subcutaneous tissue 16 centimetres in diameter was received. There was an elongated ulcer 13.5 centimetres long and varying from 3.5 to 6.5 centimetres wide. The ulcer had raised edges and ragged necrotic floor.

#### Microscopic Examination.

Skin.—Amoebae are present in parts of the floor of the ulcer.

Colon.—No amoebae were found on reexamining the original ulcer or in other sections of the colon, though there is little doubt that the lesion in the colon was due to amoebiasis.



### Summary.

1. Stricture of transverse colon due to old latent amebiasis.
2. Acute amebiasis of the anterior abdominal wall following resection of the lesion in the transverse colon.
3. Intestinal obstruction due to adhesions following laparotomy.

### Pathological Discussion.

DR. ROSE: We should learn some lessons from the presentation of this case. First, I should like to ask Mr. Wilson, who made a very accurate and practical diagnosis without committing himself pathologically, to answer any comments that have been made.

MR. WILSON: Gangrenous ulceration of the skin does occur in ulcerative colitis, but it is usually in the extremities and not near an ileostomy or colostomy. Any excoriation in those situations can be attributed directly to the irritating ileal contents. I do not agree about the absence of strictures in chronic ulcerative colitis, for they do occasionally occur by the time the patient is seen by the surgeon. As for amebiasis, I must admit I did not think the mucosa of the bowel would have been perfectly normal near an ameboma. I have seen diverticulitis localized without other evidence of its presence, and I have even seen one case with two separate masses, one in the sigmoid and one in the transverse colon, with no lesion between. In this case I certainly thought a very localized diverticulitis was more likely than amebiasis.

DR. ROSE: Should not the bacteriologist have seen these amebae? Would you care to comment on the pitfalls in diagnosing this condition, Dr. Johnston?

DR. G. A. W. JOHNSTON: The Pathology Department would have received a swabbing from the skin lesion of this patient. As there was no suggestion of amebiasis, the material on the cotton-wool swab would have been smeared and cultured. Amebae are not recognized on smears stained by Gram's method. Had the possibility of amebiasis been suggested, the correct laboratory procedure would have been to take scrapings of the skin lesion, mix with saline solution and examine microscopically. Vegetative amebae, easily recognizable as *E. histolytica*, would almost certainly have been seen in this instance.

Symbiotic gangrene is usually caused by a staphylococcus and an anaerobic streptococcus acting in combination. The isolation of a Gram-negative bacillus from this patient should have made one doubt the diagnosis of symbiotic gangrene. I would like to ask how amebae cause gangrene of the skin?

DR. TEN SELDAM: I think it is due directly to the ravages of the amebae in skin which is moist and warm, providing a suitable place for growth. I should like to support the bacteriologist's statement that it would be almost impossible to diagnose the condition by swabbing unless one was specifically asked to look for amebae, and even then it would be pretty difficult, as a dry swab is useless.

DR. CALOV: I think diagnosis is primarily a clinical matter. Unless the clinician has amebiasis in mind, the pathologist and bacteriologist have very little chance of making the diagnosis for him. One should always think of amebiasis when there is a history of a previous attack of dysentery, no matter how long ago it was. Here we have a history of two attacks.

PROFESSOR K. INGLIS: I should like to ask Dr. ten Seldam a question. We used to teach that the naked eye characteristics of the ulcers in the large intestine in amebic dysentery were different to those of bacterial cause. Would there be corresponding differences between the cutaneous condition that we have seen here and the appearance of symbiotic gangrene?

DR. TEN SELDAM: I do not think the appearance is very different. There are minor differences, but I would not like to commit myself on naked eye examination alone. I agree that typical lesions in the bowel are usually quite distinctive, but there are cases in which it is not possible to distinguish the different causes. In addition, it is not surprising that amebae were not found on reexamination of the resected portion of bowel. I remember a case in which three small ulcers were present in a caecum, which was removed under the wrong diagnosis of appendicitis, in which it was possible to find amebae in one ulcer and not in the others in spite of an intensive search, and even though there appearances were identical with the first. Similarly I have seen the bowel wall loaded with amebae, and yet during life none were found in the stools by experienced men.

PROFESSOR INGLIS: Is it common to come across gangrene in the bowel in patients with amebic dysentery?

DR. TEN SELDAM: No.

DR. ROSE: I think the success of diagnosis in these cases would be increased in this hospital if there was a greater realization on the part of clinicians that there is such a thing as surgical bacteriology. This is something I have been interested in. Tissues removed at operation go automatically to the morbid anatomy department. If they went in part to the bacteriology department, post-mortem specimens too, I think it would assist in making accurate diagnosis more often. It would have helped in this case. If there had been better liaison between different departments in the hospital, I think this patient would have been diagnosed earlier. Are there any further comments on the clinical aspects of this case?

DR. J. RIEMER: I should like to ask Mr. Wilson whether, in view of this history and of the ultimate aim of all therapy, cure of the patient, he thinks a patient should receive pre-operative preparation longer than for three days, and whether he thinks some other form of treatment would have been better advised in this case. Could a true diagnosis have been made at operation? Would the diagnosis of amebiasis have altered the line of treatment?

MR. WILSON: It does not seem possible that the diagnosis of ameboma could have been made at operation. Prior to colonic resection three days of pre-operative treatment is normally adequate when streptomycin and phthalylsulphathiazole are used, and more than adequate if neomycin is used. I think this case developed complications because of a defect in the surgical technique. There must have been some reason for doing something different from simply resecting the bowel and dropping the joined ends back into the abdomen. If there is doubt about the technique of colonic resections, it is usually whether or not the blood supply to the bowel ends is adequate. In such cases of doubt, further resection till undoubtedly viable bowel is reached is the answer. If this patient had a local lesion, an ameboma, and there was no lesion or amebae detected elsewhere, then it is likely that healing would have occurred in the usual way. He would then not have got the faecal fistula, nor the spreading destruction of the skin, nor the subsequent obstruction which caused his death.

DR. EVANS: Was this man ever outside Australia?

DR. G. MICHELL: Unfortunately he was in hospital many years ago, and there is no record of that.

### Diagnosis.

Intestinal obstruction due to adhesions and amebiasis of the colon and skin.

## British Medical Association News.

### SCIENTIFIC.

A MEETING of the South Australian Branch of the British Medical Association was held at the Repatriation General Hospital, Springbank, South Australia, on September 29, 1955, the President, Dr. G. L. BENNETT, in the chair. The meeting took the form of a series of clinical and pathological demonstrations by members of the medical, surgical and pathological staffs of the hospital.<sup>1</sup>

### Esophageal Obstruction.

DR. T. P. DEARLOVE presented details of two cases of dysphagia in patients recently admitted to hospital under his care, who, although their histories were similar, proved to have two very different conditions. They had both suffered from increasing dysphagia for about eight weeks, and for the ten days prior to their admission to hospital each had been reduced to subsisting on fluids sipped very slowly. Investigation and treatment therefore was a matter of some urgency in each case. Dr. Dearlove said that whilst it was not his intention to enumerate the various causes of dysphagia or to enter upon a discussion of the differential diagnosis, yet he would suggest to those members who might be interested in brushing up their knowledge of the subject that a perusal of the four or five relevant chapters in "Modern Trends in Gastro-Enterology" (by F. Avery Jones) would prove fruitful.

<sup>1</sup> Acknowledgement is made to the Chairman of the Repatriation Commission for permission to publish details concerning patients under the care of the department.

The first case was that of a man of fifty-one years, a "beachcomber" by profession and by no means teetotal, who gave a rather indefinite history of heart-burn on and off for the past ten years. The symptom consisted really of epigastric and lower retrosternal discomfort after drinking hot tea after cold beer. Eight weeks prior to his admission to hospital he had become aware quite suddenly of solid food and hot fluids "sticking" in the lower third of the chest region. Also during that period he had noticed pain in the same region on lying flat or bending forwards. The condition had become progressively worse. Clinical examination revealed no relevant abnormality. X-ray examination after the swallowing of barium bolus showed a constant oesophageal deformity at the site where the patient complained of the obstruction and pain.

Oesophagoscopy, performed by Dr. H. Brown, revealed a narrowing of the oesophagus at 33 centimetres, with redness but no ulceration of the oesophageal mucosa, and beyond the narrowed region could be seen gastric mucosa. Although the pathologist was unable to exclude definitely a scirrhous carcinoma from a biopsy specimen taken during oesophagoscopy, it was considered that the case was more likely to be one of herniation of the stomach through the diaphragm or of extension of gastric mucosa above the diaphragm with recurrent oesophagitis at the junction of the oesophageal and gastric mucosa with subsequent stricture—in effect, the type of condition well described by Allison. The patient was submitted to laparotomy, which was performed by Dr. H. Brown. It was found that the stomach was herniated through the diaphragm for one and a half to two inches, and there was no evidence of malignancy. After dividing the vagus nerve it was possible to reduce the herniated portion back through the diaphragm; and as the stricture had been successfully dilated, further radical treatment was considered unnecessary.

The second case was that of a sixty-three-year-old farmer, who had suffered for five to six years with chronic cough, recurrent upper respiratory tract infections, and gradually increasing shortness of breath, due to pulmonary emphysema, right lower lobe bronchiectasis and a mild degree of congestive cardiac failure. On a day six weeks prior to his admission to hospital he had been partaking of a dish consisting of chopped up and boiled sheep's head when he developed a sudden severe bursting pain in the upper central part of his chest, accompanied by the feeling that the mouthful of food just swallowed had stuck there.

The pain had persisted since that time and was aggravated by attempting to swallow anything, and only fluids could be induced to pass the site of the pain. The pain remained quite severe and radiated through to the back between the shoulder blades. An interesting point was that it was much aggravated by extending the neck. During this period the patient became very much more short of breath than usual, and, to use his own words, would "nearly strangle" if he attempted to lie flat, and would cough up copious quantities of frothy white sputum.

Examination of the patient on admission to hospital showed that he was short and thick-necked, in some respiratory distress and with a fever of 100° F. He had severe inspiratory stridor, heard best at the mouth level. The rest of the findings on clinical examination were in keeping with the previously mentioned diseases from which he suffered. There was no evidence of superior vena cava obstruction.

Dr. Dearlove said that the history, together with the inspiratory stridor, had led him to postulate that the patient must have a laryngeal or tracheal carcinoma, with involvement of the oesophagus producing partial obstruction. That proved to be incorrect. X-ray examination after the swallowing of a barium bolus produced an unusual picture. There was a constant narrowing in the region just beyond the crico-pharyngeal opening, and in the three inches of oesophagus distal to that there were three barium-filled protrusions from the posterior wall. Laryngoscopy and bronchoscopy revealed nothing abnormal. Oesophagoscopy examination was attempted by Dr. H. Brown, who found a tight stricture just beyond the crico-pharyngeal opening, which was firm and bled easily. It was possible to pass a small dilator only about one centimetre beyond that obstruction. Examination of a biopsy specimen taken from that region showed: "Agranulomatous mass associated with the presence of a fragment of dead bone, and multiple crystalloid foreign bodies of undetermined nature."

For the few days immediately following the examination the patient felt rather better and stated that he had less pain and that swallowing seemed a little easier. He looked better, and his fever was settling on treatment with antibiotics.

A few days later a further oesophagoscopy examination was performed by Dr. H. D. Sutherland at Dr. Brown's

request. On that occasion, after some rather dextrous juggling with instruments of varying calibre to negotiate the crico-pharyngeal region, where nothing grossly abnormal was to be seen, the whole length of the oesophagus was visualized. The abnormal findings were an area of granulation tissue of the left lateral oesophageal wall extending from 22 to 25 centimetres. There was no evidence of foreign body or new growth, and it was thought possible that the abnormal findings were a result of previous trauma.

The patient continued to improve, and a further X-ray examination a week later revealed an almost normal oesophagus, in which there was no evidence of the three peculiar protrusions previously present. Dr. Dearlove said that the exact nature of the protrusion remained obscure. The lowest was certainly in the region where diverticula were not uncommon, but its disappearance was unaccountable. The upper two might have represented ulceration through the oesophageal wall due to trauma, though if that was the case, the patient would appear fortunate in not having succumbed to a fulminating mediastinitis.

Dr. H. Brown discussed the differential diagnosis of the two cases presented by Dr. Dearlove. He said that in his view the diagnosis in the first case was a hiatus hernia with recurrent oesophagitis producing a benign stricture at the oesophago-gastric junction. In the second case he believed that the lesions shown in the X-ray films represented small abscesses, which he considered had been drained after the manipulation of the investigation by oesophagoscopy.

Dr. H. D. SUTHERLAND also discussed the cases. In the first case he considered that the diagnosis was one of sliding hiatus hernia, with a fibrosis spreading into the mediastinum. In the second case he agreed with Dr. Brown that the upper lesion shown in the X-ray films was due to an impacted foreign body (a spicule of bone) below the crico-pharyngeus. He thought that possibly the lower lesion was diverticulum. He remarked that it was interesting to note two cases with similar histories due to entirely different lesions.

#### Solitary Myeloma of the Femur with Pathological Fracture.

Dr. C. W. PHILLIPS presented details of a case of pathological fracture of the neck of the right femur in a man, aged fifty-six years, who had been a totally and permanently incapacitated pensioner for the previous three years because of spondylitis.

In 1917 the patient had sustained fractures of the shaft of the right femur and the shaft of the left tibia in a train accident; the fracture of the femur had been treated by the insertion of a metal "plate", which was still *in situ*. Since 1949 the patient had been treated for low back pain and pain in the right thigh, the pains being attributed to lumbar and sacro-iliac osteoarthritis. He had worn a spinal support since 1952.

During May, 1955, the right leg had become increasingly weak and heavy; despite heat and massage, the leg had become almost useless during the first week in June. On June 7 he slipped on a mat and struck his right hip on a table. Nevertheless, that minor trauma caused a fracture of the neck of the right femur. X-ray films of the right hip on his admission to hospital had demonstrated a grossly comminuted subtrochanteric fracture with detachment of the lesser trochanter; areas of translucency were present in the bone on either side of the fracture, suggesting that the fracture was a "pathological" one.

Dr. Phillips said that the use of a blade-plate to immobilize the fracture had been contraindicated by the bony changes adjacent to the fracture; treatment had been by extension in a Thomas splint. Radiological examination of the chest, spine and pelvis had revealed no abnormality, except spondylitis. The serum phosphatase levels were within normal limits. An attempt to perform a biopsy at the fracture site via a trocar and cannula had been unsuccessful. The X-ray films were examined by a radiotherapist, who suggested that the most likely diagnosis was a simple cyst, and radiotherapy was not recommended.

After ten weeks' treatment, a large amount of callus was palpable, but X-ray films revealed no evidence of union. In view of the lack of radiological evidence of union, the lesion was explored surgically by Dr. Neville Wilson. A large cyst was found and curetted; the gelatinous lining was scraped out and the cavity packed with bone chips.

Microscopic examination of the biopsy material showed a characteristic picture of myeloma. X-ray films of the skull, ribs and spine showed no abnormality and the results of repeated examination of the urine for Bence-Jones pro-



tein were negative; the results of serum protein analysis and the serum calcium level were within normal limits.

Dr. Phillips said that a diagnosis of "solitary myeloma" was considered justified, and a course of deep X-ray therapy had been instituted.

Dr. J. T. QUINLAN commented on the pathology of the lesion. He said that it was a typical myeloma histologically, and as far as could be ascertained there were no other lesions present. Sternal marrow examination had shown no abnormality.

Dr. NEVILLE WILSON discussed the case. He stressed the rapidity of clinical union, which was suggestive of a simple cyst. The lack of growth in the size of the cyst, together with the lack of union shown radiologically, had caused the exploration to be carried out. At the operation the appearances of the lesion were unusual. There was a large empty space, the tumour evidently being osteolytic. The tumour was filled with a semigelatinous material, rather like the consistency of a ripe grape, and was lined with thicker gelatinous material. He advocated deep X-ray therapy, and queried the advisability of amputation.

Dr. J. M. BONNIN asked of Dr. Quinlan whether a solitary myeloma could metastasize, or whether such cases later showed multiple lesions.

Dr. Quinlan said that cases of solitary plasmacytoma had been cured by amputation or irradiation, and in the present case amputation would be a reasonable procedure. The possibility of multiple metastasis could not be excluded.

#### Interstitial Tumour of the Testis.

Dr. J. T. QUINLAN presented details of a case in which a patient had been suffering from interstitial cell tumour of the testis (Leydig cell tumour). The patient, an obese monumental mason, aged fifty-eight years, had undergone endoscopic resection of the prostate on October 13, 1953, for prostatomegaly, from which he suffered dysuria and urinary frequency with retention. That operation relieved his symptoms only partially, if at all. After the second operation, gross enlargement of the right testis was noted. The testis ached constantly, and he felt a nagging in the right groin. It was thought that the lesion was probably infective in origin, but that neoplasm could not be excluded. On the latter ground the right testis was removed on July 30, 1954. There was no clinical or biochemical evidence of endocrine disturbance. Dr. Quinlan demonstrated the pathological specimen removed at operation, consisting of a testis, epididymis and part of the spermatic cord, the whole measuring three inches by two inches by three inches and weighing 58 grammes. Examination of the cut surface showed an irregularly lobulated brownish tumour replacing most of the testis, which was represented by a thin strip of normal tissue at the inferior margin. Microscopically the tumour was composed of very uniform polyhedral cells resembling liver cells, with a finely vacuolated and strongly eosinophilic cytoplasm. No haemorrhagic or degenerative changes were noted and only a few mitoses. The appearances seen were considered characteristic of a Leydig cell tumour.

Dr. Quinlan went on to say that although there was some uncertainty in individual prognosis, the majority of such tumours were innocent or possessed only a low grade of malignancy. He considered that the tumour demonstrated should be placed in the latter category. It was of interest that, although there had been no recurrence of the Leydig cell tumour, yet the patient had since developed an adenocarcinoma of the sigmoid colon, the latter lesion being a fungating growth about two inches long and encircling about two-thirds of the lumen of the bowel at one point. Microscopically, that bowel tumour was a well-differentiated adenocarcinoma; no local lymphatic metastases had been seen in the regional lymph nodes removed surgically.

Dr. Quinlan said that such Leydig cell tumours, or interstitial cell tumours of the testis, had been first described in 1895; Leydig had described the cells named after him in 1850. Since that time, a number of cases had been described and recognition of them had become more frequent. In children such growths tended to produce precocious puberty and macrogenitosomia. In adults, however, they were not, as a rule, associated with any endocrine disturbance, although gynecomastia had been reported in a few cases. The tumour was rare, occurring about once in every 300,000 hospital admissions. Only one previous case had been recognized in Adelaide, in a child. Dr. Quinlan related the extraordinary story that he had been told, on good authority, that that tumour had been accidentally eaten by an absent-minded physician at a clinical meeting. The tumour now presented was therefore the only one extant in South Australia.

#### Sarcoidosis.

Dr. C. D. SWAINE presented details of the case of a man suffering from sarcoidosis. The patient, a soldier, aged forty-five years, had been admitted to hospital on June 27, 1955. His annual army X-ray examination had revealed an abnormal opacity projecting into the lung field from the lower part of the right hilum and some shadows in the right lower zone. In the region of the aortic knob there was a double shadow suggesting the possibility of a glandular enlargement. At the time, the patient complained of an occasional cough with yellowish sputum, which had never been blood-stained. He had no other complaints whatsoever, and no abnormality was found on physical examination. Examination of four successive specimens of sputum failed to reveal acid-fast bacilli. Bronchoscopic examination revealed nothing abnormal, and the Mantoux reaction was negative. He was examined at the chest clinic on July 1, 1955, when a provisional diagnosis of unresolved pneumonia was made, and it was suggested that he should be discharged and reviewed in six weeks. After that interval antero-posterior and right lateral tomography showed a roundish well-circumscribed mass a little over an inch in size situated posteriorly in the lower lobe, slightly lateral to the spine. Lateral to that mass was a less well-defined, less dense smaller opacity. The radiologist considered that the appearances were suggestive of the presence of a neoplasm with a zone of collapse.

After reviewing the X-ray films and tomograms, on August 19, 1955, the chest clinic advised a diagnostic thoracotomy. Thoracotomy was performed on September 1, 1955, and the right lung was found to contain a hard craggy mass at the apex of the lower lobe and many discrete, grey pleural plaques about one-quarter of an inch in diameter. Nodules were felt within the lung substance. Examination of a frozen section of a specimen of lung tissue revealed sarcoidosis. The pathologist's report on the section read: "Section shows a tissue reaction characteristic of sarcoidosis. Acid-fast bacilli were not found and there is no caseation. Most of the newly developed fibrous tissue gives a positive reaction to stains for amyloid." Examination of the haemoglobin and a white cell count on August 30 revealed no abnormality, and a differential white cell count and blood film were normal. The serum protein content was: albumin 5.1%, globulin 2.7%, ratio 1.9:1; total protein content 7.8%. The Wassermann and Kline reactions were normal.

Dr. J. T. QUINLAN discussed the pathological and histological features of sarcoidosis, referring to its possible relationship to the features seen in cases of histoplasmosis and *periarthritis nodosa*, as well as the features of malignant granuloma. He said that the most interesting feature in the case presented, from the pathological point of view, was the early development of amyloid, for which he could offer no explanation.

Dr. R. GOLD asked Dr. Quinlan on what grounds he declared that the lesions showed the features of amyloid.

Dr. Quinlan, in reply, said that the diagnosis of amyloid was made on the characteristic staining reactions.

Dr. P. W. VESCO discussed the differential diagnosis in "unresolved pneumonia", a term that he deprecated. He said that it was often difficult to be certain about diagnosis in some cases, as the laboratory did not always help with the isolation of the tubercle bacilli. He wondered if liver biopsy had been considered in the present case, as it might possibly have been an aid to diagnosis, and might have spared the patient an operation. He asked in what proportion of such cases a positive result was obtained from liver biopsy.

Dr. Swaine, in reply, said that he was not familiar with liver biopsy in the condition under discussion, but commented on the value of scalene node biopsy—a biopsy made from the *scalenus anticus*. He thought that if sarcoidosis had been thought of in the differential diagnosis, then it might have been possible to perform such a biopsy, and so to spare the patient a major operation.

Dr. H. W. LINN stressed the importance of reactions of the reticulo-endothelial system, which could be of various types—for example, Hodgkin's disease. He referred to the phenomenon of the negative Mantoux reaction in sarcoidosis, as well as that of a changed Mantoux reaction. He referred also to some recent Scandinavian claims that a virus had been isolated from sarcoidosis patients.

Dr. R. GOLD quoted Scadding, who, he claimed, had stated that a liver biopsy was more useful than a lymph gland biopsy.

Dr. H. BROWN said that the lesions seen at operation looked like those due to tuberculosis, but against that was the fact that the Mantoux reaction was negative. He thought

that current opinion overseas was that scalene node biopsies were more likely to be of value when there were lesions in the upper pulmonary lobes, but they were a less valuable diagnostic procedure for lower lobe lesions.

DR. P. W. VESCO referred to a survey he had conducted some years before for histoplasmosis and coccidioidomycosis in South Australian cases of Mantoux-negative pulmonary disease—a survey which had yielded entirely negative results. He thought that too much stress was laid upon the fully developed radiological picture of pulmonary sarcoidosis—the “snow-storm in the chest”—and that sarcoidosis, in less obvious lesions, was commoner than was generally accepted.

DR. H. D. SUTHERLAND said that the thoracotomy was a necessary procedure in the present case, and fully justified. He referred to the surgical procedure of some American surgeons, who went to great lengths to remove a lymph gland—even a deep intrathoracic one—for biopsy by an approach through the upper part of the chest.

DR. R. C. ANGOVE discussed the value of liver biopsy. He referred to the recent work of Scadding and Sherlock, in which liver biopsy gave 100% of positive results, and also to the work of Clifford Hoyle, who had used liver biopsy as a means of checking treatment. He supported the action that the Repatriation Hospital chest clinic had taken in performing thoracotomy, saying that they had no alternative.

#### Mite Bite Dermatitis.

DR. R. V. SOUTHCOTT presented details of some cases of dermatological interest. The first was that of an orderly at the hospital, who had reported sick with a rash, mainly on the forearms, for a few inches above and below the elbows. Examination of the patient showed a number of small papules, each capped by a vesicle, and surrounded by a broad zone of erythema. Inquiry revealed that on the previous day he had been working in the guinea-pig house, where his duties included the unbalancing of straw and the putting of it to the animals. He experienced malaise for a couple of days whilst the vesicular eruption was present. Dr. Southcott said that the lesions described were typical of dermatitis from the hay itch mite (*Pyemotes ventricosus*), which examination revealed in fair numbers in the straw that the orderly had been handling. Such a dermatitis was seen in workers in chaff and straw, and was variously termed grain itch, hay itch, mattress itch, straw itch *et cetera*. The lesions seen were macroscopically similar to those seen in the scrub itch and similar itch from the bites of the Trombiculid mites. In the south-east of South Australia, along the teatree scrub bordering the Coorong, there was a “teatree itch”, seen in campers and duck shooters, due to the bite of *Trombicula samboni*, the lesions being the same as many of the audience must have experienced during military service in the tropical parts of Australia and New Guinea. In North Queensland the mite responsible was principally *Trombicula hirsti*; in New Guinea and some of the adjacent islands the mite responsible was *Schöngastia blestovici* principally. By contrast, however, the mites responsible for the transmission of scrub typhus in those regions did not cause a scrub itch. Dr. Southcott outlined briefly the life history of the hay itch mite.

DR. H. W. LINN asked if the development of the vesicles in mite-bite dermatitis was due to a sensitization phenomenon.

DR. C. M. DELAND referred to his experiences in New Guinea and the adjacent islands. He had noticed that on moving into a new area it was common for severe reactions to Trombiculid mite bites to develop. After one had remained in that area for a time, however, one appeared to become immune to the bites of the local Trombiculid mites, and no further reactions ensued. He wondered what factors were responsible for immunity to mite bite.

DR. SOUTHCOTT, in reply to Dr. Linn, said that he believed that the vesiculation produced by mite bite was due primarily to a local reaction to the toxins of the salivary secretions of the mites. In the case of the reactions to the bites of the hay itch mite, it had been suggested that sensitization reactions potentiated those effects. Dr. Southcott described the mechanism of the feeding of the Trombiculid mites, with the development of the characteristic histiosiphon or stylosome, with surrounding histolysis in the host. He referred to experiments he had conducted in 1941 with the local Trombiculid mite *Leeuwenhoekia adelaidae*, obtained from the ears of cats in Adelaide. Those mites, when partially fed on the cat, were detached and placed upon his forearm under a watchglass. They readily attached themselves to the skin by their mouthparts, and remained attached for up to three days. Vesicles developed at the site of the attachment and gradually increased in size,

becoming loculated and then coalescing, until the mite remained attached on top of a large loculated vesicle about two centimetres across on the back of his wrist. At that stage the vesicles burst, the serum unfortunately drowning the mites it was hoped to rear. By contrast, the papular dermatitis which arose from larval schistosomes, which he would next demonstrate, was due to sensitization phenomenon.

In reply to Dr. Deland, Dr. Southcott said that there was no doubt that there were big differences between the reactions to the biting of the various species of Trombiculid mites. One point of interest that he had noted was that red-headed or very fair people seemed to suffer more acutely from the bites of the North Queensland scrub itch mite (*Trombicula hirsti*) at least. He had noticed that, in a company of about 60 men in military manoeuvres in the Atherton Tableland in North Queensland, after each military exercise the same three or four men always turned up on sick parade, suffering severely from multiple mite bites. That was in the days prior to the introduction of the dimethyl and dibutyl phthalates. He had collected a good number of the scrub itch mites from those men, who were either red-haired or fair-haired. He had conducted a survey of a series of scrub typhus patients who had contracted the infection at Bramston Beach, near Innisfail. Testing if hair colour made any difference produced no apparent evidence that it made any difference to the liability to an attack of scrub typhus. It was not possible to state whether some people were more attractive to the various Trombiculid mites concerned, with a consequent greater bite rate, or whether in the case of the scrub itch the differing skin reactions were alone responsible for the differing reactions. The effects were later seen less with the introduction of the efficient mite repellents into the Australian army.

#### Schistosome Dermatitis.

DR. SOUTHCOTT next presented details of a case of “swamp itch” (or “bather’s itch”) in an infant. The patient had been examined at another institution some years before, but details of the case were presented at the meeting as it was thought that it might be of interest to members. Possibly not all of them were aware that the condition occurred in South Australia. The infant and its parents had been camping near the shallow swamps of the River Murray at Tallem Bend, South Australia. The mother had washed the infant daily at the water’s edge. After several days she noted the papular eruption, which gradually increased in severity, both in the numbers and in the size of the papules. At the time when the child was examined (January 28, 1947) some of the papules had increased to large erythematous plaques, some of which had coalesced. Dr. Southcott said that the effects were due to the penetration of the skin by the larval trematode (schistosome), believed to be *Cercaria parvella*. The rash of a schistosome dermatitis took several days to develop, and was a sensitization phenomenon, as the studies of Macfarlane and Olivier had indicated. Similar lesions occurred in Western Australia. It was believed that the flukes concerned were parasites of swans and other birds, or possibly water rats and other mammals, and that the attack on man was accidental. The intermediary host was the “bubble” snail, *Lenamaria pyramidata*, and the skin lesions demonstrated were well known in the swamps of the lower Murray. The mechanism of the penetration of the cercariae was referred to, as well as the fact that the flukes concerned could not mature in the human host. Dr. Southcott also mentioned the economic importance of the similar bathers’ itch around the Great Lakes of North America, in detracting from the tourist trade. He said that that was not at present a feature of the Australian infestations, but it might one day become so. Possible preventive measures were mentioned, including the use of copper salts and other molluscicides.

#### Xanthoma Tuberosum.

DR. SOUTHCOTT’S next case was that of a press operator, aged forty-one years, who had been admitted to hospital for a course of intramuscular heparin therapy. He had had military service in the Northern Territory, New Britain, Morotai and Borneo. The history at the time of admission to hospital (March, 1954) was only of four years’ duration. At the onset he had noticed the appearance of small nodules on his elbows. Those had gradually increased in size and had tended to coalesce. Small nodules had also appeared over his *tendo calcaneus* on each side, and on his knees, and those also had gradually enlarged. Over the six months prior to admission to hospital further nodules had developed over the buttocks, and in the palms of the hands. The nodules on the elbows, knees and heels caused him no concern, except by their increase in size and on being



knocked. Those on the buttocks tended to become sore as a result of sitting. The small nodules which had appeared under the skin of the palm were extremely tender, and interfered with his work, at working handles, levers *et cetera*. There was nothing of significance in his previous medical history, and the medical history of the family was also non-contributory. His wife and three children were well. He had two younger siblings; they were unaffected, and his parents were living and unaffected. There was no known Jewish strain in his family history.

On examination he was noted to be of middle height, rather heavily built and blue-eyed. Each eye had a well-marked *arcus senilis*, which went right around the cornea. The nodules were present as described in the history. In each of them there was a nodular collection of yellow material under the skin in the situations as recorded. In places where the lesions tended to be rather exposed to trauma, for example, the elbows and heels, the skin over the lesions was reddened, but was intact. In the palms the lesions were about the size of a grain of wheat, or a little smaller. In the little fingers there was a small thickened plaque (the size of a sago grain) of the same yellowish material underlying the flexure of the first interphalangeal joint. The lesions in the palms were all tender. Clinical examination of the patient revealed no abnormality in heart, lungs, nervous system or other systems, and the blood pressure was normal. The Wassermann and Kline reactions were negative, the blood hemoglobin value, white cell count and differential white cell count were normal. The electrocardiogram was normal. The serum cholesterol level was greatly raised, being estimated at repeated examinations (on admission and at intervals over the four weeks whilst he was an in-patient) at values ranging from 820 to 930 milligrammes per 100 millilitres. He was given a course of heparin by intramuscular injection in a dosage of 2000 units twice weekly over one month, as well as a diet of low cholesterol content. There was no response to the therapy, in either the character of the lesions or the level of the serum cholesterol. He had been treated since that time at the out-patient clinic by the reduction of cholesterol intake and by thyroid therapy. However, he did not appear to have benefited from any treatment, and his blood cholesterol level had remained high.

Dr. Southcott discussed briefly the classification of the lipoidoses, referring particularly to the xanthomatoses. He said that the patient under discussion had no evidence of previous diabetes, chronic pancreatitis or nephritis, or of familial idiopathic hyperlipemia, and his disease could be classified in the group of primary xanthomatoses. Such cases could be divided into two groups, as had been done, for example, by Robb-Smith: (a) Those with a raised blood cholesterol level. They constituted a varying group, ranging from the xanthelasmas of the eyelids to tuberoses xanthomas at the other end of the scale, in which latter category he placed the lesions of the patient shown. They were frequently familial. In diagnosis they might be mistaken for psoriasis or gout. Frequently they were associated with major cardio-vascular involvement. (b) The normocholesterolaemic. In those red-brown skin lesions occurred, which were more widely disseminated than in the previous group.

Dr. H. W. LINN discussed the differential diagnosis of the xanthomatoses, referring to the classification of Thannhauser. He referred to the role of the lipoids in that condition.

A MEETING of the Victorian Branch of the British Medical Association was held at the Repatriation General Hospital, Heidelberg West, on July 6, 1955. The meeting took the form of a series of clinical demonstrations by members of the medical and surgical staffs of the hospital.

#### Hæmochromatosis.

DR. G. R. WIGLEY and DR. W. E. KING presented details of six cases of hæmochromatosis illustrating the various facets and the modes of presentation of this condition. In four cases the diagnosis had been proved by means of liver biopsy, and in two cases the clinical diagnosis had been obvious. Two cases had been predominantly diabetic in presentation, two had presented as obscure hepatomegaly, and one each had presented with pigmentation and with endocrine hypofunction. Two patients had given proven family histories of the condition.

"Kodachrome" slides and histological sections were exhibited to illustrate the findings from liver and gastric biopsies, and post-mortem specimens also were shown with the typical macroscopic appearances of the liver, pancreas,

heart and lymph nodes. The specimen of liver showed also the presence of a hepatoma *in situ*.

#### Peripheral Nerve Lesions.

DR. A. C. SCHWIEGER and DR. A. R. GILCHRIST showed four patients with neurological conditions.

A man, aged sixty-three years, had a history of progressive wasting and weakness of the limbs over the past eight years. The symptoms had been observed initially in the lower limbs, where the weakness and wasting were confined to muscles below the knees. Shortly after those effects became apparent a similar state became apparent in the upper limbs distally, the weakness and wasting gradually extending proximally. At no time did the patient complain of sensory changes, of pain or of sphincter involvement. Examination of the patient revealed atrophic weakness of all four limbs, which was profound distally. All tendon reflexes were absent. During the course of development of the condition occasional fasciculation of muscle had been observed, particularly in the early stages. No sensory changes were apparent, the muscles were not tender, and no thickening of peripheral nerves had taken place. Muscle irritability was not present. The cerebro-spinal fluid was normal in all respects, and the Wassermann reaction was negative. Histological examination of a specimen of nerve and muscle disclosed evidence of a degenerative process of some standing, in which the muscle changes appeared to be secondary to changes in the nerve. Electromyography showed complete absence of electrical activity in distal muscles; in the more proximal groups a considerable reduction in the number of action potentials was found, even to 10% of normal in muscles in which weakness was only just detectable clinically. Action potentials which were present were of an abnormal pattern. In the discussion of the differential diagnosis four conditions were mentioned—progressive muscular atrophy, myopathy, peroneal muscular atrophy and chronic motor neuropathy. It was shown that the bulk of the evidence favoured the diagnosis of true progressive muscular atrophy.

A man, aged thirty-six years, stated that he had been born with "curved fingers". However, his hands functioned perfectly normally until he suffered from severe diphtheritic paralysis in 1940. During his convalescence he noticed that both his hands were becoming weak, and soon he found that wasting had taken place. He had no other complaint until it was pointed out to him during the course of an examination that he was unable to feel pin-pricks on the upper portion of his body. Since then he had been aware of a difference in his appreciation of warmth over the affected area as compared with elsewhere. He denied any disturbance of sphincter control and claimed that his lower limbs were normal. Examination of the patient revealed a loss of corneal reflexes together with analgesia and thermoanesthesia in the presence of intact light touch sensation over the entire trigeminal territory on both sides. Otherwise cranial nerve function was intact. Examination of the upper limbs revealed a curious contracture of the fingers. The interphalangeal joints were fixed, and the flexor tendons were prominent in the palm. The patient said that that had been present for as long as he could recall. In addition, however, there was obvious wasting of the small hand muscles with gross weakness. Power elsewhere was normal, and the reflexes were brisk. In the lower limbs the only detectable weakness was in hip flexion, where it was slight. The patient's reflexes were brisk; and whilst neither plantar reflex was of frank extensor type, both were abnormal. In addition, the abdominal reflexes became fatigued readily. On the sensory side there was analgesia to pinprick with partial thermoanesthesia over the trunk and upper limbs from the ninth dorsal segment upwards to where it became continuous with the trigeminal areas. Light touch was preserved, as were position sense and two-point discrimination. The condition was considered to be syringomyelia, with dissociated sensory loss from involvement of the spinothalamic tracts from the ninth dorsal segment to the spinal tract and nucleus of the trigeminal nerve, as well as evidence of lower motor involvement in the upper limbs and a pyramidal lesion affecting the lower limbs, together with a congenital deformity.

A man, aged twenty-nine years, had given a history that four months prior to his seeking advice he had experienced difficulty in replacing an electric light globe with either of his arms. He said that he had had no prior disability, and questioning revealed nothing relevant in the family history. Examination of the patient showed features suggestive of a myopathic facies. He had weakness of the *orbicularis oculi* and *orbicularis oris* muscles, and in smiling he showed weakness of the retractors of the angles of the mouth. His upper limbs were well developed with two exceptions: the

muscles of the posterior axillary folds were very wasted, and there was severe winging of the scapulae. The only other abnormalities present were slight weakness of dorsiflexion of the ankles and the presence of a high arch in the feet. The condition was considered to be one of facio-scapulo-humeral muscle dystrophy.

In the last case considered there was some doubt concerning the diagnosis. The patient was a man, aged sixty-five years. Eighteen years previously he had noticed that his hands were becoming wasted and weak. He had had no obvious sensory symptoms or pain. Examination of the nervous system showed no abnormalities other than gross wasting and weakness of the small muscles of both hands and of the flexors of the forearms and fingers. Full investigation had been carried out over recent years. The cerebrospinal fluid was normal. X-ray examination of the cervical part of the spine showed evidence of mild spondylitis with minimal encroachment on the intervertebral foramina between the fifth and sixth and the sixth and seventh cervical vertebrae; myelography showed only slight evidence of "median bar" formation at those levels. Radiologically the thoracic outlet was normal. On a single occasion a weakly positive Wassermann reaction had been recorded. It was considered that the picture might represent a variety of syphilitic amyotrophy.

#### Leucaemia.

DR. JOHN BOLTON and DR. R. H. D. BEAN discussed the treatment of leucaemia with cytotoxic drugs. Particular attention was given to the treatment of chronic lymphatic leucaemia and chronic myeloid leucaemia with triethylene melamine and "Myleran" respectively. The indications were discussed for small frequent doses and for larger doses at long intervals, and illustrative case histories were presented. The value was emphasized of supportive treatment and in particular of a diet high in first-class protein content and supplemented by intramuscular administration of vitamins; and it was pointed out that in several cases the duration of remissions appeared to have been lengthened by the addition of such a regime to the treatment. Complications were then discussed, and the practical importance was emphasized of recognizing as a predominantly hypersplenic phase a sudden fall in the peripheral blood count occurring shortly after the drug had been taken. The dramatic results obtained by treating this phase with antibiotics, blood transfusion and cortisone were demonstrated.

#### Digitalis Intoxication and the Electrocardiogram.

DR. J. J. HURLEY discussed some of the lesser known aspects of digitalis intoxication, especially auricular arrhythmics. He said that those included auricular premature beats, which were frequently blocked, and auricular tachycardia with either dissociation or block. The latter form of arrhythmia occurred commonly and was almost specific for digitalis intoxication. The auricular rate varied between 120 and 250 per minute and was associated with varying degrees of heart block. The P-R interval varied. A Wenckebach phenomenon might be present with occasional dropped beats, and a 2:1 block might develop. Loss of potassium potentiated the toxic action of digitalis. That might occur as a direct result of the pathological process itself or as a result of treatment involving electrolyte manipulation—for example, an unpalatable diet or a diet of low sodium content, treatment with such drugs as "Mersalyl" and "Diamox", or loss of electrolytes in vomiting or diarrhoea. Such loss of intracellular potassium, the serum potassium content remaining normal, might precipitate the characteristic arrhythmia. Treatment with potassium chloride and "Pronesty" was illustrated and discussed. A warning was issued against rapid digitalization in the aged and in those with badly damaged hearts.

#### Primary and Secondary Myxoedema.

DR. A. BARDSEY and DR. F. CATARINICH showed three patients suffering from primary hypothyroidism and two patients with hypopituitarism.

Dr. Bardsey discussed the modes of presentation of hypothyroidism with particular reference to the patients shown, emphasizing the anaemia, rheumatic manifestations, various psychogenic disorders, symptoms referable to the ear, nose and throat, and cardio-vascular changes. He stressed the importance of the changes in the voice and played recordings of patients' voices before and after treatment.

Dr. Catarinich discussed the differential diagnosis between primary and secondary myxoedema and the importance and dangers of ill-conceived therapy or investigation. He pointed out that no single factor made the diagnosis certain; rather was it made on the over-all pattern of history, signs and investigation.

#### Ulcerative Colitis.

DR. H. A. PHILLIPS and DR. LINDSAY GRIGG showed two patients suffering from ulcerative colitis who had undergone a two-stage total colectomy. They illustrated the fact that it was unwise to bury the sigmoid stump with a view to subsequent perineal excision. That had been found to hold a grave risk of leaving a segment of sigmoid behind and also of damaging the bladder and its nerve supply. It was advised that the proximal end of the recto-sigmoid remnant should be exteriorized in the left flank and that subsequent resection should be by an abdomino-perineal approach, when the excised bowel was clearly under vision at all times.

#### Lacerated Rectum.

Dr. Phillips and Dr. Grigg also showed a young soldier, who had been impaled on a stake and had lacerated his rectum and anal sphincters. He had been treated by sigmoid colostomy and cleaning of the perineum with delayed suture of the sphincters and subsequent closure of the colostomy. Emphasis was laid upon the importance of laying the wound track widely open at the initial operation, the good result in the present case having been substantially due to that practice.

#### Orthopaedic Demonstration.

DR. F. V. STONHAM and DR. J. H. GLADSTONE presented details of five cases of tuberculosis of the spine to demonstrate unusual lesions and difficulties in treatment, as well as methods of grafting the spinal column. They also displayed a number of photographs of menisci removed from the knee, showing typical lesions, arthrograms and the histological features of the chronic chondritis which occurs in such cases.

#### Dupuytren's Contracture.

DR. JOHN HUESTON presented illustrative patients and clinical photographs to demonstrate the condition of Dupuytren's contracture. Apart from the classical palmar bands and flexion deformity of the condition, other clinical forms were presented. They included: (a) involvement of the natatory (web) ligament, producing limitation of finger abduction; (b) continuity of the little finger band with the deep fascia of the *abductor digiti minimi* with rapidly progressing deformity and a poor prognosis; (c) involvement of the dorsal knuckle pads with cosmetic deformity; (d) involvement of the plantar fascia with bilateral lesions.

The histological picture of the condition was presented, and the danger was stressed of interpreting the sometimes pleomorphic hypercellular fibrous tissue as fibrosarcoma. That had at first been the histological diagnosis made from one of the nodules demonstrated from the plantar fascia. Hence it was necessary to remember the possibility that this innocent condition could occur in the foot. Operation was advised only in the presence of disability, which might be of two types: (a) pain arising from pressure on fascial nodules or from the stretching of fascial bands and (b) flexion deformity sufficient to interfere with work or sport.

Simple fasciotomy might suffice to relieve disability in the elderly patient with discrete mature bands. Fasciectomy was advised for the progressing condition associated with disability, a regional rather than a total excision being favoured. Secondary joint changes, if they could not be corrected by fasciectomy to a useful range, justified amputation. Brachial plexus block was favoured, as bleeding was reduced on release of the tourniquet.

#### Colectomy for Ulcerative Colitis.

DR. ROBERT LAWSON and DR. LINDSAY GRIGG showed a man, aged thirty years, who had suffered from ulcerative colitis for ten years, having spent as long as eighteen months continuously in hospital. X-ray examination after the administration of a barium enema showed the characteristic pipe-stem appearance of the colon. The patient was having from eight to 12 motions daily, with passage of blood and mucus, and had lost three stone in weight. In December, 1954, colectomy was performed, the terminal part of the ileum being brought out of a McBurney incision and the distal terminal end of the sigmoid colon being brought out through a similar left-sided incision. Convalescence was uneventful, the patient leaving hospital in eighteen days. After six months the patient was completely transformed in health, had gained three stone in weight, was working hard as a production engineer and was full of vigour and energy and able to swim, dance and enjoy heavyweight boxing. The ileostomy gave little trouble, draining continuously into a disposable bag without leakage or skin irritation. The opening of the terminal end of the sigmoid colon remained almost dry, being only a quarter of an inch in diameter, while a little mucus (but no blood) was passed *per rectum*.



every second day. An X-ray examination of the recto-sigmoid loop after injection of an opaque medium showed that it was narrowed to a diameter of about half an inch, and that part of the bowel was evidently fibrotic and quiescent. Resection of that terminal segment was not contemplated.

#### Recurrent Sigmoid Volvulus and Resection.

Dr. Lawson and Dr. Grigg also showed a man, aged fifty years, who in May, 1954, had been found on examination to have an acute sigmoid volvulus with gross abdominal distension and wide fluid levels due to obstruction visible on X-ray examination of the abdomen. At operation the torsion of the sigmoid colon was untwisted and the bowel was deflated by means of a tube passed *per rectum*. The patient made a smooth recovery, and the matter of resection of the colon was deferred. In April, 1955, the patient was again admitted to hospital with a recurrence of acute sigmoid volvulus. After the sigmoid loop had been untwisted and the bowel deflated as previously, the redundant length of colon was brought out of a left-sided McBurney incision, and the ends were cut off. Subsequently the spur of the colostomy so formed was crushed with an enterotome, and the colostomy was finally closed six weeks after the original operation. At the time of the meeting the patient was in full health with normal bowel function and sound healing of his wounds.

#### Recurrent Parotid Tumour with Subtotal Parotidectomy.

The third patient shown by Dr. Lawson and Dr. Grigg was a woman, aged thirty years, who had had local removal of a parotid tumour in 1944, 1952 and 1953. Deep X-ray therapy had been given and radon implantation had been performed after the third operation. When she had come under the care of her present medical attendants in October, 1954, she appeared to have a further local recurrent nodule. There was a good deal of induration and scarring from the previous operation and irradiation. In November, 1954, subtotal parotidectomy was undertaken, the branches of the facial nerve being dissected forwards from the trunk at the stylo-mastoid foramen. Very slight facial weakness occurred after the operation, but photographs taken three months after the operation showed full restoration of all facial movements with a smooth and inconspicuous scar at the operation area.

#### Carcinoma of the Stomach: Prostatomegaly.

The last patient shown by Dr. Lawson and Dr. Grigg was a man, aged seventy-six years, who had had a history of epigastric fullness and discomfort for two months. X-ray examination after a barium meal revealed a large filling defect in the mid-gastric area. At operation in May, 1955, a large mobile fungating tumour was found arising from a broad base on the anterior stomach wall. Partial gastrectomy was performed. The tumour on examination proved to be a papilliferous type of carcinoma. During convalescence from the operation retention of urine occurred, and prostatectomy was required. At the time of the meeting the patient had made a good recovery from both operations.

#### Urological Demonstration.

Dr. D. B. DUFFY presented a series of X-ray films to show pathological conditions of the lower end of the ureter. They included calculus, ureteroceles, hydroureter and traumatic conditions following gynaecological operations.

#### Low Back Pain.

Dr. J. C. McNEUR tabulated the causes of low back pain and illustrated them with a series of X-ray films and pathological specimens. He laid emphasis on postural, metabolic and congenital defects, which were not so commonly accepted as being the cause of backache, as opposed to the well-recognized intervertebral disk lesion. He said that postural backache was characterized by a pain in relation to standing and by a sharp lumbo-sacral angle with an abnormally horizontal sacrum. Metabolic causes were most commonly found in middle-aged to elderly females with excessive dorsal kyphosis and lumbar lordosis in association with radiological evidence of general osteoporosis. Dr. McNeur emphasized the place of active exercises and manipulations in the treatment of all affected backs, apart from those in which the trouble was due to tuberculosis, osteomyelitis, tumour and ankylosing spondylitis, and particularly in the presence of disk lesions. He said that that form of therapy had been found to produce much more lasting and complete improvement than the use of rigid supports, such as plaster jackets and spinal braces. In a certain number of cases operative treatment for prolapsed intervertebral disks was necessary. In cases of osteoporosis it was important to insist on a diet of high protein content to build up the

deficient protein matrix of the bones; when combined with the vitamin B complex and with male and female hormones, it often produced dramatic improvement. For spondylolisthesis the most satisfactory treatment was excision of the loose posterior fragment.

#### Arthrodesis of Joints.

Dr. W. R. GAYTON and Dr. D. L. GUNDRY presented a series of patients who had undergone successful arthrodesis of various types involving the hip, the knee and the ankle joint.

A man, aged twenty-five years, had presented himself in February, 1952, with a six months' history of pain in the right hip and limitation of movement. He had wasting of the gluteal and thigh muscles and loss of weight. X-ray examination showed evidence of destructive arthritis, and a presumptive diagnosis of tuberculous hip was made, which was confirmed by subsequent progress and investigation. He was treated with bilateral leg traction in Thomas splints. In April, 1952, he was placed in a plaster bed with skin traction. The disease remained clinically and radiologically active and progressive until October, 1952; at that stage X-ray examination showed evidence of improvement, and his condition began to settle down clinically. Thereafter he progressively improved, until March, 1953, when arthrodesis of the hip was performed after and during treatment with streptomycin and PAS. A modified Brittain arthrodesis was performed, with subtrochanteric osteotomy and tibial bone grafting. A nail and plate were used in addition to the Brittain operation. After operation he was nursed in a plaster bed, and chemotherapy was continued. In July, X-ray examination suggested that arthrodesis of the hip joint was occurring. In August, X-ray examination showed that arthrodesis was established, and that fusion between the femur and the graft was occurring. There was no evidence of union of the inner end of the graft, which was evidently in the obturator foramen. At that stage the patient was taken out of plaster and allowed to progress very gradually to weight-bearing. In October, X-ray examination showed further obliteration of the joint line. Union between the graft and femur was sound. Weight-bearing was continued with the help of crutches. When he was discharged from hospital, he was well, and the right hip was painless. Since then he had been reviewed as an out-patient. His hip ankylosis had remained solid, and there had been no evidence of activity.

A male patient had presented in November, 1954, with the complaint of pain in both hips since 1945, steadily increasing in severity since 1951. Movement in the hip was severely limited. The pain in the right hip had become so severe for the six months prior to the patient's admission to hospital that he was unable to carry on his business. Examination revealed severe muscle wasting around the right hip with flexion deformity of 35° to 40°. Movement was limited to a few degrees only. Arthrodesis of the right hip was advised, to be followed later by arthroplasty of the left hip, if necessary. In December, 1954, Brittain's "V" arthrodesis of the right hip was carried out, with insertion of a tibial bone graft of large dimensions. The hip was placed in a plaster spica. After operation some discomfort was experienced from the hip spica, but otherwise convalescence was remarkable for the rapid improvement in general health and comfort of the patient consequent on the relief of his pain. He also gained weight. In March, 1955, X-ray examination showed that fusion of the graft and probably fusion of the joint were commencing. A short spica was applied, and mobilizing exercises were commenced. In May, 1955, the short spica was removed, and the patient became ambulant.

Another patient had polyarthritides of a mixed type, chiefly rheumatoid. The right knee had progressively become more painful and swollen, and 30° of flexion deformity had developed with only 20° to 30° of painful flexion available. No response had resulted from medical treatment including butazolidin and later cortisone. Finally arthrodesis was advised. This was carried out by contact compression. The joint surfaces were excised, and the flexion deformity was corrected. The joint was found to show severe osteoarthritic changes. After operation the patient was nursed in a Thomas splint, and convalescence was comfortable. Five weeks after operation clinical evidence of arthrodesis was present, and seven to eight weeks after operation the patient was allowed to become ambulant in a caliper. At a four months' review, when he was an out-patient, the knee was found to be painless, and clinical and radiological union was satisfactory.

A man, aged sixty-five years, had suffered a gunshot wound of the right thigh and an injury to the right knee in the 1914-1918 war. His complaint on the present occasion was of a painful right knee for many years with severe

restriction of movement. Examination of the right knee revealed the presence of an effusion, varus deformity and a 10° flexion deformity. Flexion was limited to 40° of movement, and that movement was slow and painful. X-ray examination showed the presence of gross osteoarthritis. At operation in July, 1954, arthrodesis of the right knee joint was carried out by contact compression. After operation the patient was nursed in a Thomas splint with the Steinmann pins resting on the splint. Two days after operation excessive extension and some varus deformity were noticed in check X-ray films. Both of these were corrected easily by alteration of the splint *in situ*. Convalescence was uneventful, and in accordance with the claims for this method the patient was relatively comfortable. Nursing management was without difficulty. On August 19 the rubber bands were removed, and the union was tested. It was found to be clinically firm. The leg was lifted out of the splint supported only at the heel. On September 6 the Steinmann pins were removed, and on September 20 mobilization in a caliper commenced. When the patient was discharged from hospital, the right knee was painless; and review films at the end of five months showed firm X-ray union with trabeculation across the fracture site. The patient was advised to retain his caliper for protection.

A man who had suffered a fractured left ankle in 1917 sought advice in 1953 for a four-year-old complaint of intermittent sharp pain and constant dull ache in the ankle. He was found to have swelling and deformity of the ankle. All movements were limited, but not severely so. On December 7, 1953, arthrodesis of the ankle was carried out with excision of the joint surfaces and insertion of a chip graft. A below-knee padded plaster was applied. On February 25, 1954, X-ray examination showed progress of the arthrodesis; the plaster was reapplied with a walking iron. On April 8 well-advanced union was seen, the plaster cast was discarded, and mobilization was commenced. On April 22 the patient's ankle was painless, but he was walking with a limp. The heel was built up a quarter of an inch on the inside. On September 12 his ankle was painless, and he was walking well.

The last patient had suffered a Pott's fracture in 1945. In November, 1954, he complained of increasing pain and stiffness in the left ankle with considerable pain on walking. He was unable to wear normal footwear. Examination of the patient revealed one inch of wasting of the left calf with deformity of the ankle, and tenderness over and anterior to the medial malleolus. Ankle movement was grossly restricted. X-ray examination showed gross osteoarthritic changes. On February 14, 1955, arthrodesis of the left ankle was performed, the tibial, talar and fibular joint surfaces being excised. The bone obtained was milled and packed in as a graft. No other form of graft was used. A below-knee padded plaster was applied. On February 28 the sutures were removed, excessive equinus was corrected, and the plaster was reapplied. On March 17 the patient was discharged from hospital on crutches. On April 28 X-ray examination showed that union was proceeding. On July 4, 1955, X-ray examination showed further consolidation of the arthrodesis.

(To be continued.)

## Out of the Past.

In this column will be published from time to time extracts, taken from medical journals, newspapers, official and historical records, diaries and so on, dealing with events connected with the early medical history of Australia.

### WHAT WE LEARN FOR PLEASURE IN PROSPERITY WE MUST TURN TO PROFIT IN ADVERSITY.<sup>1</sup>

[From the *Sydney Gazette*, 18 July, 1818.]

MR GEORGE WHITE M.I.A. Dentist and Physician respectfully offers his services to the Gentry and Public at large of Sydney: wishing to inculcate in their minds the necessity of an early attention to the teeth of their children. The neglect of removing teeth of an extraneous growth disarranges the entire set and renders them unhappy through

<sup>1</sup> From the original in the Mitchell Library, Sydney.

life. To Adults he recommends immediate application to get their teeth cleaned from that calcareous matter that does indescribable mischief. Mr White makes one tooth to an entire set, equal to nature: whereby mastication and articulation are effected with improved clearness—A certain malady cured with certainty secrecy and expedition. The poor attended to each day until eleven o'clock (Sunday excepted). Tooth powder of a superior quality. Specimens of Teeth may be seen at his residence in Phillip St. Sydney.

## Correspondence.

### FEEs AND THE GENERAL PRACTITIONER.

SIR: At a meeting of the Association in Brisbane in April, 1956, it was resolved that fees in general practice are inadequate, but a base line was difficult to set. Actuarial investigation would justify our claims, but it could not do more than calculate hours and patients against income; no allowance could be made for being on call, correspondence, telephone disturbances in recreation, study and meal times, and their effect on our health and families. A three-man practice here calculated a profit of 1s. 10d., approximately 2s. 9d. net taxable income per patient, or about 12s. an hour over all.

The belief common to most of us is that already we expend more time and mental effort for our money than any tradesman and most other professionals; so the exhortation to work harder to compensate for an unstable national economy is impossible to obey unless we shamelessly overtear. Morally unacceptable and physically impracticable. Already it is necessary to process 30 or more at 15s. in three hours to maintain our income, without too frequent rushed examinations followed by needless reference for costly investigations and return visits.

We consider the resolutions of the general practitioners' meeting of June, 1955, in Brisbane to impose no real hardship on the public, calling for an increase only in charges for after-hours services (6 p.m. to 8 a.m.), and lengthy consultations—namely, £2 2s. visits and £1 1s. consultations, anything after 6 p.m. being practically 8 p.m., coming at mealtime. The figure was calculated on the time taken to treat most genuine emergencies as one hour out and back, and the strong feeling that after ten hours at ordinary rates we were due for something like "time and a half on time", still not considering "waiting time" for being available at any hour as too difficult to assess, and part of the job. A few of the more indignant members who were seeing their families as visitors adhered to these fees, and still our nights are spoiled by routine examinations to the same number as before.

We have been forced to continue regular night hours to keep our patients from going where these are available, since management will not allow time off for medical attention when they know doctors have always worked at night. Except in exceptional circumstances, this should not be—it is possible to attend for ancillary investigations as well as effect all business transactions during the day. Only the local general practitioner is commanded to work at night. While accepting the ideals of the profession, we feel these should not include unfair conditions, as implied in the belief that the doctor is for the service of the people. What indignities have we general practitioners suffered by the omission of "sick" to qualify "people".

To provide a full service it is necessary to arrange a relief for any time off in a seven-day week, and work twice as hard while he has his due rest. No worker is allowed to accept such conditions without adequate recompense.

Our members have an early demise in general, with little chance to save except by life insurance, which is not available unless the coronary attack is fatal rather than crippling; in the latter event all income ceases. We should like more security for our families.

We were warned that the Government will not permit too great a discrepancy between fees and benefits, and has prevented the funds from increasing present refunds. This is fixing fees on a schedule drawn up three years ago, before surgery and domestic expenses soared. If a controlled investigation shows a justification for a rise in fees, an adjustment must be forced by the public, instructed if necessary at our expense to the factors determining such a rise.

Any small rise would not appreciably affect the position we are in financially, as it would be swallowed up in taxes,



incite bureaucratic reprisals, and without lengthy and expensive public relations appeals upset our honest patients. They might, however, make it worthwhile to chase confirmed non-payers, who consider medical benefits an unprofitable investment, while accounting for £300 to £400 annually in small amounts, in this practice serving housing settlements.

Yours, etc.,

771 Old Cleveland Road,  
Camp Hill,  
Brisbane.  
May 5, 1956.

J. K. FARNWORTH.

#### APPENDICITIS IN NEW GUINEA NATIVES.

Sir: Dr. Wilson's factual report of two genuine cases of appendicitis among New Guinea natives interests me. From 1935 to 1938 I was resident in the Persian Gulf, and the consensus of opinion amongst the American mission doctors was that appendicitis was unknown in the true Bedouin Arab, who lived on dates and dried fish. However, with modern-day oil interests, the Bedouin Arab was becoming a town Arab, eating meat and using fat in his cooking. Appendicitis was fairly common amongst town Arabs.

I note that both the cases quoted by Dr. Wilson were in natives living under civilized conditions and probably eating food cooked in fat. Possibly a civilized person seldom has enough physical exercise during his average working day to completely break down fat to carbon dioxide and water. While it is at the stage of acetone and diacetic acid fat seems to favour the onset of disease.

Yours, etc.,

Perth,  
Western Australia,  
May 3, 1956.

S. C. S. COOKE.

### Naval, Military and Air Force.

#### APPOINTMENTS.

THE undermentioned appointments, changes *et cetera* have been promulgated in the *Commonwealth of Australia Gazette*, Number 17, of April 19, 1956.

#### NAVAL FORCES OF THE COMMONWEALTH.

##### Permanent Naval Forces of the Commonwealth (Sea-Going Forces).

###### Retired List.

**District Naval Medical Officer.**—Surgeon Commander Godfrey Joseph Kelleher Lane is appointed District Naval Medical Officer, New South Wales, dated 12th March, 1956.

##### Citizen Naval Forces of the Commonwealth.

###### Royal Australian Naval Volunteer Reserve.

**Termination of Appointment.**—The appointment of Archibald Gordon Murray as Surgeon Lieutenant is terminated, dated 1st February, 1956.

#### AUSTRALIAN MILITARY FORCES.

##### Citizen Military Forces.

###### Northern Command.

**Royal Australian Army Medical Corps (Medical).**—The provisional rank of F1/1017 Captain M. R. Edye is confirmed. 1/39181 Captain (provisionally) H. H. Moy relinquishes the provisional rank of Captain and is transferred to the Reserve of Officers (Royal Australian Army Medical Corps (Medical)) (Northern Command) in the honorary rank of Captain, 15th February, 1956.

###### Eastern Command.

**Royal Australian Army Medical Corps (Medical).**—2/147950 Major C. E. M. Gunther is appointed from the Reserve of Officers, and to be Temporary Lieutenant-Colonel, 1st February, 1956. To be Captain (provisionally), 29th February, 1956: 2/146608 Con Scott Hathaway Reed.

###### Southern Command.

**Royal Australian Army Medical Corps (Medical).**—To be Captain (provisionally), 29th February, 1956: F3/1030 Alison Kinnear Garven.

###### Western Command.

**Royal Australian Army Medical Corps (Medical).**—5/26525 Captain C. J. Benson is seconded whilst in the United Kingdom, 22nd January, 1956.

##### Reserve Citizen Military Forces.

###### Royal Australian Army Medical Corps.

**Northern Command.**—To be Honorary Captains: Anthony James Jephson Emmett, 24th February, 1956, and Raymond William Hyde Bailey and Brian Vincent Burke, 27th February, 1956.

**Eastern Command.**—To be Honorary Captain, 7th March, 1956: William Courtenay Marshall.

**Southern Command.**—To be Honorary Captain, 6th February, 1956: Alan Douglas Callister.

**Central Command.**—To be Honorary Captain, 16th February, 1956: Ruth Lorna Dow.

#### ROYAL AUSTRALIAN AIR FORCE.

##### Permanent Air Force: Medical Branch.

The following Flight Lieutenants are transferred from the Reserve and appointed to a short-service commission on probation for a period of twelve months: Peter John Bayliss (036611), 7th January, 1956; Warren Joseph Bishop (0210441), 13th February, 1956.

Henry William McKenna (0217686) is appointed to a short-service commission, on probation for a period of twelve months, 25th January, 1956, with the rank of Flight Lieutenant.

The probationary appointment of Flight Lieutenant W. H. Koschade (0311673) is confirmed.

The following officers are appointed to a permanent commission, 20th October, 1955: Squadron Leaders D. B. Heylan (023063), D. B. G. Baillie (039232), Flight Lieutenant (Acting Squadron Leader) M. A. May (013705).

Flying Officer A. E. Greentree (022681) is promoted to the rank of Flight Lieutenant, 13th October, 1955.

The resignation of Flight Lieutenant L. N. Walsh (039386) is accepted, 14th December, 1955.

##### Active Citizen Air Force: Medical Branch.

No. 22 (*City of Sydney*) Squadron.—Flight Lieutenant A. W. Raymond (025592) is transferred to the Reserve, 5th January, 1956.

No. 23 (*City of Brisbane*) Squadron.—Sidney William Page (015293) is appointed to a commission, 25th February, 1956, with the rank of Flight Lieutenant.

No. 25 (*City of Perth*) Squadron.—The following are appointed to a commission, 23rd September, 1955, with the rank of Flight Lieutenant: John Andrew Rogers (051378), Joseph McDonald Quinlivan (051379).

**Adelaide University Squadron.**—Flying Officer (Temporary Flight Lieutenant) H. D. Kennare (04900) is transferred from the General Duties Branch and is promoted to the rank of Flight Lieutenant, 7th October, 1955. Pilot Officer L. S. Coats (04733) is transferred from the Reserve, 31st October, 1955.

##### Air Force Reserve: Medical Branch.

The following former officer is appointed to a commission, 26th September, 1955, with the rank of Flight Lieutenant (Temporary Squadron Leader): J. R. McCoy (253345).

Air Cadet Redford John Wright-Smith (036614) is appointed to a commission, 16th December, 1955, with the rank of Flight Lieutenant.

The following are appointed to a commission with rank as indicated: (Flight Lieutenant (Temporary Squadron Leader)) John Douglas Hicks (257943), 28th October, 1955; (Flight Lieutenant) Ian Gordon Nicol (431736), 11th August, 1955; Robert Anthony MacMahon (268055), 13th August, 1955; John Wentworth Shand (0211529), 5th November, 1955; Keith Dudley Coulthurst (433005), Boyd Lionel Hilton Leigh (268054), 6th November, 1955.

The following Air Cadets are appointed to a commission, provisionally, 25th January, 1956, with the rank of Pilot

Officer: Maxwell Walter Swingler (036641), Arthur Victor Leslie Hill (036613).

The provisional appointment of the following Pilot Officers is confirmed and they are promoted to the rank of Flight Lieutenant: T. M. McGrath (015153), 10th December, 1954; E. W. Pick (034797), 15th December, 1954; R. G. Cameron (036604), 19th December, 1955.

The name in the notification regarding the appointment of Nerine Brustolin (257939) to a commission, as approved in Executive Council Minute No. 2 of 1956, appearing in *Gazette* 8, dated 16th February, 1956, is amended to read: "Nerine Brustolin."

The appointment of Flight Lieutenant W. K. Collins (5911) is terminated, 3rd February, 1956.

## Royal Australasian College of Surgeons.

### PRIMARY EXAMINATION FOR THE FELLOWSHIP.

A PRIMARY EXAMINATION in anatomy (including normal histology) and applied physiology and the principles of pathology for Fellowship of the Royal Australasian College of Surgeons will be conducted in Melbourne, Sydney and Dunedin in September, 1956.

Written papers will be held simultaneously in the three cities on Thursday and Friday, September 6 and 7, 1956. The examiners will visit the three centres for the purpose of conducting the viva-voce section of the examination.

The examination is reciprocal with primary examinations for Fellowship of the Royal College of Surgeons of England, the Royal College of Surgeons of Edinburgh, the Royal College of Surgeons in Ireland and the Royal Faculty of Physicians and Surgeons of Glasgow.

Each examination is open to graduates of not less than one year's standing of a medical school approved by the Council of the College for the purpose.

Candidates must submit evidence of their qualification and of the date of acquirement thereof.

Forms of application for admission to the examination may be obtained from the Secretary, Royal Australasian College of Surgeons, Spring Street, Melbourne.

When entering for the examination, candidates must state whether they desire to appear before the Board of Examiners in Melbourne, Sydney or Dunedin.

The fee for admission or readmission to the examination is £15 15s. (plus exchange on cheques drawn on banks outside Melbourne). The fee must be forwarded with the form of application so as to reach the secretary at his office in Melbourne not later than July 26, 1956.

It is stressed that entries close at the College office in Melbourne on July 26, 1956, and that late entries cannot be accepted.

## Post-Graduate Work.

### THE POST-GRADUATE COMMITTEE IN MEDICINE IN THE UNIVERSITY OF SYDNEY.

#### Week-End Course at Broken Hill.

THE Post-Graduate Committee in Medicine in the University of Sydney, in conjunction with the Broken Hill Medical Association, will hold a week-end course at the Broken Hill and District Hospital on Saturday, June 2, 1956. The programme is as follows:

Saturday, June 2: 9.30 a.m., "Some Principles in the Management of Emphysema", Dr. Cotter Harvey; 10.45 a.m., "Asthma from the Psychiatric Side", Dr. David Ross; 11.45 a.m., "Atopic Eczematitis", Dr. J. C. Bellisario; 2 p.m., "Miliary Conditions of the Lungs", Dr. Cotter Harvey; 3 p.m., "Modern Therapeutics", Dr. J. C. Bellisario; 4.15 p.m., "The Integration of Psychiatry in Internal Medicine", Dr. David Ross.

The fee for attendance at the course will be £5 5s., and those wishing to attend are requested to communicate as soon as possible with Dr. F. Schlink, Honorary Secretary, Broken Hill Medical Association, 252 Mica Street, Broken Hill. Telephone: Broken Hill 865.

### DISEASES NOTIFIED IN EACH STATE AND TERRITORY OF AUSTRALIA FOR THE WEEK ENDED MAY 5, 1956.<sup>1</sup>

Disease.	New South Wales.	Victoria.	Queensland.	South Australia.	Western Australia.	Tasmania.	Northern Territory.	Australian Capital Territory.	Australia.
Acute Rheumatism .. ..	..	2(1)	1(1)	..	..	..	..	..	3
Amoebiasis .. ..	..	..	..	..	..	..	..	..	..
Anoxytoma .. ..	1	..	..	..	..	..	..	..	1
Anthrax .. ..	..	..	..	..	..	..	..	..	..
Bilharziasis .. ..	..	..	..	..	..	..	..	..	..
Brucellosis .. ..	..	2(1)	..	1	..	..	..	..	3
Cholera .. ..	..	..	..	..	..	..	..	..	..
Chorea (St. Vitus) .. ..	..	3(1)	..	..	..	..	..	..	3
Dengue .. ..	..	..	..	..	..	..	..	..	..
Diarrhoea (Infantile) .. ..	4(3)	6(3)	..	..	4(4)	..	1	..	18
Diphtheria .. ..	8(5)	4(4)	3	..	2(1)	..	..	..	17
Dysentery (Bacillary) .. ..	..	3(2)	3	..	1(1)	..	..	..	7
Encephalitis .. ..	..	1(1)	..	..	..	..	..	..	1
Filariasis .. ..	..	..	..	..	..	..	..	..	..
Homologous Serum Jaundice .. ..	..	..	..	..	..	..	..	..	..
Hydatid .. ..	57(34)	69(23)	..	10(6)	7(3)	1	2	..	136
Lead Poisoning .. ..	..	..	1	..	..	..	..	..	1
Leprosy .. ..	..	..	..	..	..	..	..	..	..
Leptospirosis .. ..	3	..	20(2)	..	..	..	..	..	23
Malaria .. ..	..	..	..	..	1(1)	..	..	..	1
Meningococcal Infection .. ..	1	3(2)	..	..	..	..	..	..	4
Ophthalmia .. ..	..	..	..	..	1	..	..	..	1
Ornithosis .. ..	..	..	..	..	..	..	..	..	..
Paratyphoid .. ..	..	..	..	..	..	..	..	..	..
Plague .. ..	..	..	..	..	..	..	..	..	..
Poliomyelitis .. ..	8(4)	12(3)	4(1)	..	2(1)	..	..	..	26
Puerperal Fever .. ..	1(1)	..	..	2(2)	1(1)	1	..	..	3
Rubella .. ..	..	14(12)	..	..	..	..	..	..	17
Salmonella Infection .. ..	..	..	..	..	..	..	..	1	1
Scarlet Fever .. ..	18(9)	27(10)	7(2)	8(2)	4(3)	1(1)	..	..	66
Smallpox .. ..	..	..	..	..	..	..	..	..	..
Tetanus .. ..	..	1(1)	..	..	..	..	..	..	1
Trachoma .. ..	..	..	..	..	3	..	13	..	16
Trichinosis .. ..	..	..	..	..	..	..	..	..	..
Tuberculosis .. ..	23(18)	18(14)	5(3)	2(2)	20(16)	7(5)	..	..	75
Typhoid Fever .. ..	1(1)	..	..	..	..	..	..	..	1
Typhus (Flea-, Mite- and Tick-borne) .. ..	..	..	..	..	1	..	..	..	1
Typhus (Louse-borne) .. ..	..	..	..	..	..	..	..	..	..
Yellow Fever .. ..	..	..	..	..	..	..	..	..	..

<sup>1</sup> Figures in parentheses are those for the metropolitan area.



## The Royal Australasian College of Physicians.

### GRANTS FROM THE RESEARCH FUND.

THE Royal Australasian College of Physicians invites medical practitioners to submit applications for grants from the research fund of the College. Grants may be made for research fellowships tenable in Australia or overseas or as grants-in-aid in approved circumstances. Inquiries should be addressed to the Honorary Secretary of the College at 145 Macquarie Street, Sydney.

## Notice.

### INTERNATIONAL CONGRESS.

ONE of the objects of the Council for International Organizations of Medical Sciences (CIOMS) is to coordinate as far as possible international congresses of medical and related sciences. To achieve this coordination and avoid undesirable overlap, congress organizers are requested to inform the CIOMS of their plans before fixing the dates of their meeting. The address is CIOMS, UNESCO House, 19 Avenue Kléber, Paris 16, France.

## Australian Medical Board Proceedings.

### TASMANIA.

THE following have been registered pursuant to the provisions of the *Medical Act, 1918*, of Tasmania, as duly qualified medical practitioners: Halcombe, Maureen Ena, M.B., B.S., 1955 (Univ. Sydney); Annetts, David Lyle, M.B., B.S., 1955 (Univ. Sydney); Pearce, John Warren, M.B., B.S., 1955 (Univ. Sydney); Matar, James Henry, M.B., B.S., 1956 (Univ. Melbourne); Lang, John Keith, M.B., B.S., 1956 (Univ. Sydney); Lang, Marie Elizabeth, M.B., B.S., 1956 (Univ. Sydney).

## Corrigendum.

AN error appears in the letter by Dr. W. J. McCristal on the subject of "Coronary Disease and Myocardial Infarction" which appeared in the issue of April 28, 1956, at page 722. The error is on page 723 in the second line of the last paragraph but one of Dr. McCristal's letter. The last word but one in the line, which appears as "lip", should be "lipid". We regret this error.

## Medical Appointments.

Dr. J. Allison-Levick has been appointed a medical officer in the Mental Hygiene Branch of the Department of Health, Victoria.

Dr. Bertram James Phillips has been appointed to the School Medical Service in the Department of Public Health, New South Wales.

Dr. Phyllis Edna Rodriguez has been appointed Registrar to the Radiotherapy Department at the Royal Adelaide Hospital, South Australia.

Dr. G. C. Young has been appointed Psychiatrist Superintendent, Mental Hygiene Branch, Department of Health, Victoria.

## Nominations and Elections.

THE undermentioned have been elected as members of the New South Wales Branch of the British Medical Association: Cummings, Royal, M.B., B.S., 1956 (Univ. Sydney); Fay, Warwick Harvey, M.B., B.S., 1956 (Univ. Sydney); Stack, Ellen Mary, M.B., B.S., 1956 (Univ. Sydney); Dawson, Peter Edgar Rowland, M.B., B.S., 1953 (Univ. Sydney); Ferguson, William John, M.B., B.S., 1952 (Univ. Sydney); Lane, Godfrey Joseph, M.B., B.S., 1932 (Univ. Melbourne); Rec, Otakar, licensed under Section 21(b) of the *Medical Practitioners Act, 1938-1955*.

THE undermentioned have been elected as members of the South Australian Branch of the British Medical Association: Barker, Denis, M.B., B.S., 1955 (Univ. Adelaide); McLeay, Leslie Margaret, M.B., Ch.B., 1936 (Univ. Manchester).

## Diary for the Month.

MAY 31.—South Australian Branch, B.M.A.: Scientific Meeting.  
MAY 31.—New South Wales Branch, B.M.A.: Branch Meeting.  
JUNE 1.—Queensland Branch, B.M.A.: General Meeting.  
JUNE 5.—New South Wales Branch, B.M.A.: Organization and Science Committee.  
JUNE 5.—Victorian Branch, B.M.A.: Branch Meeting.  
JUNE 6.—Western Australian Branch, B.M.A.: Branch Council.  
JUNE 8.—Tasmanian Branch, B.M.A.: Branch Council.  
JUNE 8.—Queensland Branch, B.M.A.: Council Meeting.

## Medical Appointments: Important Notice.

MEDICAL PRACTITIONERS are requested not to apply for any appointment mentioned below without having first communicated with the Honorary Secretary of the Branch concerned, or with the Medical Secretary of the British Medical Association, Tavistock Square, London, W.C.1.

**New South Wales Branch** (Medical Secretary, 135 Macquarie Street, Sydney): All contract practice appointments in New South Wales.

**Queensland Branch** (Honorary Secretary, B.M.A. House, 225 Wickham Terrace, Brisbane, B17): Bundaberg Medical Institute. Members accepting LODGE appointments and those desiring to accept appointments to any COUNTRY HOSPITAL or position outside Australia are advised, in their own interests, to submit a copy of their Agreement to the Council before signing.

**South Australian Branch** (Honorary Secretary, 80 Brougham Place, North Adelaide): All contract practice appointments in South Australia.

**Western Australian Branch** (Honorary Secretary, 8 King's Park, West Perth): Norseman Hospital: all contract practice appointments in Western Australia. All government appointments with the exception of those of the Department of Public Health.

## Editorial Notices.

MANUSCRIPTS forwarded to the office of this journal cannot under any circumstances be returned. Original articles forwarded for publication are understood to be offered to THE MEDICAL JOURNAL OF AUSTRALIA alone, unless the contrary be stated.

All communications should be addressed to the Editor, THE MEDICAL JOURNAL OF AUSTRALIA, The Printing House, Seamer Street, Glebe, New South Wales. (Telephones: MW 2651-2-3.)

Members and subscribers are requested to notify the Manager, THE MEDICAL JOURNAL OF AUSTRALIA, Seamer Street, Glebe, New South Wales, without delay, of any irregularity in the delivery of this journal. The management cannot accept any responsibility or recognize any claim arising out of non-receipt of journals unless such notification is received within one month.

**SUBSCRIPTION RATES.**—Medical students and others not receiving THE MEDICAL JOURNAL OF AUSTRALIA in virtue of membership of the Branches of the British Medical Association in the Commonwealth can become subscribers to the journal by applying to the Manager or through the usual agents and book-sellers. Subscriptions can commence at the beginning of any quarter and are renewable on December 31. The rate is £5 per annum within Australia and the British Commonwealth of Nations, and £6 10s. per annum within America and foreign countries, payable in advance.